TO STUDY THE PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION, 24TH AND 48TH HOUR

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Dedicated

to

my family

&

Teachers

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Dr. Shubham Goyal

LIST OF ABBREVIATIONS

AP	:	Acute pancreatitis
PLR	:	Platelet to lymphocyte ratio
NLR	:	Neutrophil to lymphocyte ratio
ERCP	:	Endoscopic retrograde cholangiopancreatography
MOF	:	Multiorgan failure
MODS	:	Multiorgan dysfunction syndrome
SIRS	:	Systemic inflammatory response syndrome
MPV	:	Mean platelet volume
POF	:	Persistent organ failure
CRP	:	C-reactive protein
ICU	:	Intensive care unit

ABSTRACT

Introduction: Acute pancreatitis is defined as an acute condition presenting with abdominal pain, a three-fold or greater rise in serum levels of pancreatic enzymes amylase or lipase and/or characteristic findings of pancreatic inflammation on imaging methods (including abdominal ultrasonography and computed tomography).

Aim of Study: To study the efficiency of platelet to lymphocyte ratio as marker to predict the outcome of acute pancreatitis.

Materials & methods: This prospective study will include patients presenting with acute pancreatitis in Dayanand medical college and hospital between 1st march,2021 to 28th feb,2022. Platelet to lymphocyte ratio will be calculated as ratio of absolute platelet count to absolute lymphocyte count at the time of presentation to hospital and at 24th and 48th hour of presentation to hospital. Mean of PLR value at time of presentation, 24th and 48th hour of presentation to hospital will be calculated. Patients will be divided into 3 groups according to mean platelet to lymphocyte ratio of < 150 as IST group, 150-300 as 2nd group and > 300 as 3rd group and length of hospital stay, need of ICU/HDU stay, DOR/death/DAMA/discharge, type of management whether (medical, PCD, surgical), organ failure occurrence/MODS, complications of acute pancreatitis will be compared between the groups. The results will be statistically analysed.

Results: We observed that increase in PLR >300 was significantly associated with poor prognosis while ≤150 was associated with better prognosis with respect to organ failure, complications of Acute pancreatitis, need for ICU and outcome of patient.

Conclusion: Each scoring system has its advantages and disadvantages. The PLR has advantages over other scoring systems such as being easy-to-use, simple, highly sensitive and specific and providing information regarding mortality and prognosis without requiring 48 hours of assessment time.

Keywords: Acute pancreatitis (AP), Platelet to Lymphocyte ratio (PLR)

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INTRODUCTION

Acute Pancreatitis (AP) is defined as an acute condition manifesting with abdominal pain, a threefold or greater increase in the serum levels of the pancreatic enzymes amylase or lipase, and/or characteristic findings of pancreatic inflammation on imaging modalities such as computed tomography and abdominal ultrasonography.[1,2] It is an inflammatory process characterized by the activation of cytokine cascades in response to local pancreatic damage, resulting in systemic inflammation.[3] It is one of the most prevalent disorders of the digestive tract, resulting in a significant emotional, physical, and financial burden on the human population. [4] In affluent nations, the incidence of acute pancreatitis ranges from 4.9 to 35 per 100,000 people. Annually, acute pancreatitis causes 327,474 hospitalizations and 2,834 deaths in the United States.[5] Acute pancreatitis is most prevalent between the fifth and sixth decades; nonetheless, mortality increases with age. As alcohol use and biliary calculi are the two most common causes of acute pancreatitis, it has been hypothesized that there are regional and socioeconomic disparities in the incidence of the disease.[6]

AP is the consequence of a complicated process with varying etiology and natural history. The most prevalent causes of AP are gallstones and alcohol intake; however, hypertriglyceridemia, medications, endoscopic retrograde cholangiopancreatography (ERCP), trauma, obesity, diabetes, and infection are all well-known causes of local and systemic inflammation.[6,7] Other risk factors for acute pancreatitis include undiagnosed genetic variants, exposure to smoking and other environmental pollutants, and the effects of concomitant disorders.[8] Acute pancreatitis is a paradigmatic model defined by local and systemic inflammation, which is recognized clinically from the absence of systemic symptoms through the systemic inflammatory response syndrome, organ failure, prolonged organ failure, and death.[1,9] Patients with elevated

platelet-to-lymphocyte ratios are more likely to be admitted to the intensive care unit, to have longer hospital stays, and to die. Consequently, elevated PLR readings correlate with a poor outcome in acute pancreatitis. [1]

The clinical manifestations of acute pancreatitis may be generic and have a low predictive sensitivity (40%) for adverse outcomes.[1] In some autopsy series, previously undetected acute pancreatitis was shown to be the cause of death, demonstrating that there is a high risk of underdiagnosis. [5] In AP, early mortality is caused by SIRS, resulting in MODS, but late mortality is typically caused by sepsis and its consequences.[10] Although the case fatality rate for AP has reduced over time, the population mortality rate for AP has not changed. [4]

Two separate AP stages have been identified:

I. Early phase (within one week) is marked by the systemic inflammatory response syndrome (SIRS) and/or organ failure.
II. Late phase (> 1 week) marked by local problems.[4]

It is crucial to acknowledge the utmost significance of organ failure in defining the severity of a disease. [4] Local complications are defined as peripancreatic fluid collections, sterile or infected pancreatic and peripancreatic necrosis, pseudocysts, and walled-off necrosis (sterile or infected). [4]

The underlying pathophysiology of the progression of local pancreatic injury to SIRS is not fully understood.[3] The progression from onset of Acute Pancreatitis to Severe Acute Pancreatitis is driven by an inflammatory cascade, which is initiated by TLR-nuclear factor kB (NF kB) activation and cytokine production in acinar cells.[11,12] Inevitably, severe and persistent SIRS results in multiple organ failure determines the character of the systemic inflammatory response is the extent to which these mediators escape into the circulation. Lastly, pancreatic infection may recur if the inflammation does not resolve.[13] Previous research

indicates that somatostatin (SST) has a strong anti-inflammatory effect on AP. Our group's basic study revealed that octreotide, an equivalent of SST, might disrupt the TLR4-NF kB-cytokine pathway, reduce the activation of intestinal mucosal mast cells, and enhance B-cell maturation in macaques.[11,14]

Due to the various manifestations of AP and it's unclear pathogenesis, early diagnosis and treatment of the disease, as well as a precise risk assessment to classify patients as high or low risk, and admission of patients with organ dysfunction to intensive care units, are crucial. [4] Due to the potentially deceptive presentation of AP, different severity rating systems have been created to assist doctors in triaging patients who will likely require intensive care unit therapy (ICU).[5] These scoring systems should, ideally, employ predictive tools that are speedy, reproducible, affordable, minimally invasive, and highly accurate. [5,15]

Different rating systems have been developed to evaluate severity and predict prognosis. Balthazar and the early warning score, which utilize CT and physical exam findings, respectively, are examples.[5] The others are the Ranson score, the APACHE II score, the Bedside Index of Severity in Acute Pancreatitis (BISAP) score, and the Glasgow-Imri criteria.[1] The acute physiologic assessment and chronic health evaluation (apache) and Ranson criteria are the most common scoring techniques.[11] These scoring systems are time-consuming, utilize numerous factors that are difficult to gather in situations with limited resources, and are complex.[1]Several Inflammatory indicators, such as C reactive protein (CRP), procalcitonin, IL-6, and IL-8, have been demonstrated to accurately predict the early diagnosis of Systemic Inflammatory response syndrome (SIRS), however they are expensive and difficult to get.[1,16]

Numerous research groups have explored the predictive relevance of the hematological components of the systemic inflammatory response for disease severity and outcome during the past decade. We still require

simple, effective, and quick markers to predict the severity of acute pancreatitis prognosis in the present day.[9] The neutrophil to lymphocyte ratio (NLR), red cell distribution width (RDW), mean platelet volume, and platelet to lymphocyte ratio can be obtained using inexpensive laboratory procedures. [9] In various chronic inflammatory illnesses, accumulating evidence suggests that Mean Platelet Volume plays a significant role as a measure of inflammation, disease activity, and anti-inflammatory treatment efficacy.[17] Platelet activating factor is primarily secreted by vascular endothelium and is a pro-inflammatory mediator that causes localized tissue damage and bleeding in autoimmune polyarthritis. Platelets and neutrophils are activated by PAF, which also enhances capillary permeability.[17] Platelets are a source of inflammatory mediators and are regulated by arterial surface contact. The mediators are released by activated platelets; platelet adhesion and its atherothrombotic potential might then cause the release of mediators and the continuation of the inflammatory process.[18]

The ratio of platelets to lymphocytes has emerged as an informative metric that reveals changes in platelet to lymphocyte numbers related to acute inflammatory and prothrombotic conditions. In neoplastic disorders accompanied by immunosuppression and thrombosis, the platelet-to-lymphocyte ratio, which can be predicted by total blood cell counts and their ratios, has been thoroughly explored.[19] PLR has been identified as an inflammatory marker, and platelets have been identified as active participants in antimicrobial host defense, induction of inflammation, and tissue repair.[1] Platelet counts correspond with platelet reactivity and volume and may indicate autoimmune disease activity, anti-inflammatory therapy response, and the presence of numerous comorbidities.[20,21,22] In vitro and in vivo studies have elucidated the origin of activated circulating platelets, which exhibit numerous membrane receptors and release numerous biologically active substances from their granules. These

platelets can withstand cellular interactions and immune, inflammatory, and thrombotic diseases. [23,24,25]

At the locations of vascular injury, circulating platelets can interact with erythrocytes, neutrophils, and lymphocytes in the artery lumen.[26,27] The interaction of platelets with t lymphocytes, mediated by p- selectin, reduces lymphocyte proliferation, leading to a decrease in proinflammatory cytokines, such as interferon- alpha (IFN- ALPHA), tumor necrosis factor-alpha (TNF ALPHA), and interleukin (IL-17), and an increase in anti-inflammatory cytokines, such as IL-10. Transient lymphopenia and the magnitude of stress-induced hypercortisolemia with subsequent platelet release influence the degree of increase of PLR in numerous proinflammatory and prothrombotic illness conditions. This non-specific mechanism of PLR elevation can be inhibited by increased platelet breakdown or consumption at sites of immunological inflammation and thrombosis, necessitating cross-checks of all blood cell counts and other inflammatory and immune indicators.[29]

Therefore, we propose a study to search the efficiency of platelet to lymphocyte ratio in predicting the severity of acute pancreatitis at admission, 24^{th} and 48^{th} hour.

AIM & OBJECTIVES

To study the efficiency of platelet to lymphocyte ratio as marker to predict the outcome of acute pancreatitis.

REVIEW OF LITERATURE

Interactions between blood cells are crucial to the pathophysiology of inflammation, immunological responses, coagulation, and oncogenesis. These interactions are complex, and it is frequently challenging to identify the major initiating signals and the exact functions of each cell type in the onset and progression of disease conditions. 19 The steady-state megakaryocytopoiesis process contributes to the circulation of blood by creating disc-shaped anucleate particles with a size range of 1-2 m and a half-life of 8-10 days. [17] They are referred to as platelets. They are pieces of megakaryocyte cytoplasm. 30 Platelets have a rather consistent shape throughout their lifespan. Many hormonal and immunological factors affect the maturation of thrombopoietic cells and their discharge into the circulation. Important among them are thrombopoietin, granulocyte-macrophage colony-stimulating factor, interleukin (IL)-1, tumour necrosis factor (TNF)-alpha, and interleukin (IL)-6. 17 The regulation of platelet function and aging is dependent on the ploidy (ability to reduplicate DNA) and maturity of thrombopoietic progenitors in order to maintain platelet mass and prolonged haemostatic potential. [31]

Platelets are the first cellular corpuscles that gather at a site of arterial wall injury and in the surrounding tissue. Platelets develop new receptors for adhesive proteins in response to stimulation, aggregating and releasing a multitude of cytokines and other mediators.[32] Platelets that have been activated interact directly with endothelial cells, granulocytes, monocytes, and lymphocytes. By means of these interactions, platelets can either stimulate or inhibit the functioning of other cellular components in

inflammation, and they cause the trans endothelial migration of leukocytes into the surrounding tissue.[32] Platelets are rich in proinflammatory chemicals and capable of releasing highly active microparticles; they are also intricately engaged in the development and maintenance of a variety of inflammatory illnesses.[19] Platelet counts correlate with platelet volume and reactivity and may be indicative of autoimmune disease activity, anti-inflammatory therapy response, and the presence of other comorbidities.[19] Platelets are a source of inflammatory mediators, and their contact with the arterial surface influences them. Platelet Activating Factor (PAF), a proinflammatory mediator primarily secreted by the vascular endothelium that contributes to local tissue damage and bleeding, is of key importance for inflammation in AP. PAF stimulates platelets and neutrophils, increases capillary permeability, and leads to the development of hypovolemia and edema.[33] The activated platelets release the mediators; subsequently, platelet adhesion and its atherothrombotic potential might lead to the release of mediators, the advancement of the inflammatory process, and the propagation of intracoronary thrombus that predisposes to thrombotic events.[18]

Brief anatomy of pancreas:

It is a retroperitoneal organ without a capsule. About 80 grams is its weight. It is composed of a head, a body, a tail, and an uncinate process. Bile duct's distal end passes through the pancreatic head and connects with the pancreatic duct as it enters the duodenum. The celiac and superior mesenteric arteries, which carry blood to the abdominal organs, nourish the pancreas. The pancreas is supplied with venous drainage through the splenic and superior mesenteric veins, which drain into the portal vein.[34,35,36]

Acute pancreatitis (AP) is pancreatic inflammation. AP is caused by a number of reasons. Gallstones are the most common culprit. [8] Migrating gallstones temporarily obstruct the pancreatic duct, a mechanism common

with other known causes (such as endoscopic retrograde cholangiopancreatography [ERCP]) and hypothesized causes (i.e., pancreas divisum and sphincter of Oddi dysfunction). Acute pancreatitis is not associated with malfunction of the pancreatic sphincter of Oddi or the pancreas divisum, according to controlled studies. Alcohol is the second most common cause of acute pancreatitis. Alcohol-related pancreatitis requires long-term exposure (four to five drinks per day for more than five years), and heavy drinkers have a 2 to 5% lifetime risk of getting the illness. Men are at a greater risk than women, presumably due to differences in alcohol consumption or genetic background.[37] The complex mechanisms by which alcohol causes acute (or chronic) pancreatitis involve both direct toxicity and immunologic mechanisms.[38] A number of medications, including azathioprine, 6-mercaptopurine, didanosine, valproic acid, angiotensin-converting-enzyme inhibitors, and mesalamine, have been related to acute pancreatitis, but they appear to be responsible for only a tiny percentage of cases.[39] Frequently, drug-induced pancreatitis is mild.[40] Several gene mutations and polymorphisms have been associated with acute (and chronic) pancreatitis, including mutations in the genes encoding serine protease inhibitor Kazal type 1 (SPINK1), cationic trypsinogen (PRSS1), cystic fibrosis transmembrane conductance regulator (CFTR), calcium-sensing receptor claudin-2, and chymotrypsin C.[37] These variations may interact synergistically with one or both of the risk factors for acute pancreatitis. Obesity and diabetes are also risk factors for acute pancreatitis, chronic pancreatitis, and pancreatic cancer.

The severity of acute pancreatitis (AP) can range from moderate inflammation to severe destruction of the pancreas. AP is consistently associated with a systemic inflammatory response syndrome (SIRS) in all its phases, which is triggered by a local process of pancreatic and peripancreatic autodigestion.[41] This disorder's most severe manifestation is severe acute pancreatitis (SAP). Significant rates of morbidity and mortality are associated with SAP. SAP offers a course with two phases.

During the first one to two weeks, systemic inflammatory response syndrome is caused by pro-inflammatory response (SIRS). If SIRS is severe, there may be an early onset of multisystem organ failure (MOF). After the first one to two weeks, the patient is at risk for intestinal flora translocation and the development of secondary infection of the necrotic tissue, which can lead to sepsis and late MOF.[42]

Due to a lack of understanding of the complicated immunological events that occur during AP including immune cells and inflammatory chemicals, the management of the severe type of AP is limited.[1] The mechanism that initiates AP is the activation of trypsinogen in the pancreas. This is hypothesized to be the result of excessive ductal pressures (as in the case of duct obstruction) as well as calcium balance and pH abnormalities. Because calcium transport is an ATP-driven process, particularly for sequestration in the smooth endoplasmic reticulum, it is believed that many toxins responsible for pancreatitis (including alcohol) lead to ATP depletion, resulting in elevated intra-acinar calcium concentrations that stimulate the early activation of trypsinogen to trypsin, which activates enzymes such as elastase and phospholipase. Early activation of these zymogens results in localized tissue damage and the release of DAMPs. The synthesis of DAMPs causes the recruitment of neutrophils and the initiation of the inflammatory cascade. This inflammatory cascade is responsible for the systemic symptoms of acute pancreatitis and can ultimately culminate in multiorgan dysfunction syndrome (MODS), which is the major cause of morbidity and mortality in acute pancreatitis. It is believed that elevated levels of inflammatory cytokines in the environment are responsible for systemic symptoms and consequences.[33] Following the initial stress, lymphocytes proliferate and subsequently drop during the first 24 hours, resulting in lymphopenia. Coagulation abnormalities and variations in platelet count have been found to be connected with the severity of the disease in AP.[33]

In the context of systemic inflammation, the activation and regulation of neutrophils and platelets play a vital role in developing host defences. Platelet to lymphocyte ratio (PLR), which may be determined from a full blood count, is an unique index that combines the predictive qualities of an individual's platelet and lymphocyte count to reflect a systemic inflammatory burden. [43]

Increased ratios have been reported to be connected with a variety of cardiovascular ailments and other diseases, such as colorectal malignant diseases, obstructive apnoea syndrome, essential hypertension, diabetes mellitus, and inflammatory diseases.[44] Moreover, previous research has revealed that PLR is associated with increased in-hospital mortality.[44] Elevated PLR ratios have been linked to inflammatory diseases, and poor outcomes in severe AP can be attributed to uncontrolled SIRS and its progression into multiorgan dysfunction syndrome.[3] The combination of a high platelet-to-lymphocyte ratio and an elevated platelet count may aid in the diagnosis of some systemic vascular disorders, such as giant cell arteritis (Arme yuri Gasparyan et al). In acute pancreatitis, changes in platelet count and coagulation abnormalities have been linked to illness severity.[45]

Mimidis K, Papadopoulos V, Kotsianidis J et al (2004)45 This study focused on two sequential endpoints: (1) the activation of platelets during acute pancreatitis and (2) the changes in platelet number and indices between disease start and remission, which represent the bone marrow response. The enrolment of a group of 54 patients with acute pancreatitis. The disease's cause and severity (APACHE II score) were estimated. Using flow cytometry, the Active Platelet Ratio (APR) was calculated at onset and remission. During beginning and remission, platelet count (PLT), mean platelet volume (MPV), platelet large cell ratio (P-LCR), and platelet distribution width (PDW) were measured. The results of the current investigation revealed that the first endpoint was met in patient 14,

whose APR was raised at the onset of acute pancreatitis (p = 0.01). The second endpoint was met in patient 12 for MPV, P-LCR, and PDW, which were enhanced during disease remission (p 0.01), but not for PLT (p = 0.34) until the final patient. Platelets are directly involved in the systemic inflammatory process of acute pancreatitis, which leads to consumption and an early bone marrow reaction, found the study.

Cognasse F et al. (2007)[23] analysed the role of blood platelets in CD40/CD40L-dependent immunological responses and the interactions between blood platelets and peripheral B cells. Using flow cytometry and confocal scanning laser microscopy, the scientists analysed the ability of platelets to bind non-stimulated B cells and phenotypic alterations. Modulation of cytokines/chemokines and total levels of immunoglobulin (Ig) A, IgG, IgM, and IgG subclasses in supernatants of coculture, platelets, and B lymphocytes was accomplished by sandwich enzyme-linked immunosorbent assay and differential production of cytokine messenger RNA as determined by reverse transcriptase polymerase chain reaction. Platelets and B lymphocytes were cooperatively activated in coculture, as evidenced by the enhanced expression of platelet CD62p and B-cell CD86. Platelet/B-cell interactions were accompanied by alterations in CD40 and CD40L membrane expression by both platelets and B lymphocytes. The reduction in IL12p70 and IL8 gene transcription was mainly related to B cells. In contrast, platelet-dependent reductions in sCD40L and RANTES mRNA expression were observed. Differentiated B cells enhanced their in vitro production of IgG1, IgG2, and IgG3 after a 3-day incubation with platelets, but not IgG4, IgA, or IgM. These findings highlighted the possible significance of platelets in the adaptive immune response. Platelets have an immunoregulatory function that may be utilized in multitransfused patients.

Kefeli A et al (2014)[46] examined whether MPV values would be useful as a prediction factor for patients with AP. This study involved a total of 140

AP patients (male/female: 63/77) and 70 healthy volunteers (male/female: 23/47) Age, gender, platelet count, and the mean platelet volume (MPV) were taken from hospital medical records at the time of admission and on the first day of disease remission. Mean platelet volume levels at the onset and remission of AP were 7.8±1.6% and 7.8±0.9%, respectively, with no statistically significant difference between the two groups. Platelet counts at beginning and remission were 203± 74× 103/l, 234±76×103/l, and 251±87×103/l, respectively, and there was a statistically significant difference between the two groups. At both the onset and remission of AP, the platelet count was statistically lower than in control patients. The authors concluded that MPV might be utilized as an indicator of AP and a predictive factor for AP, but the results of this investigation indicate that MPV values do not differ between AP and controls. Hence, additional prospective studies examining the factors influencing platelet size are required to evaluate whether MPV has a clinical significance and for AP patient prognostic value.

Ilhan M et al. (2015)[47] determined if neutrophil to lymphocyte ratio (NLR), platelet to lymphocyte ratio (PLR), and red blood cell distribution width (RDW) to platelet ratio (RPR) inflammatory markers could be used as reliable markers for early prediction of Acute Pancreatitis in pregnancy and if there is a correlation between disease severity and these markers. The study group consisted of 14 pregnant women who developed AP, while the control group comprised 30 pregnant women who were healthy. The NLR, PLR, and RPR were determined for both groups. NLR was significantly elevated in the AP group compared to the control group ($p=0.00$), whereas there were no statistically significant differences in PLR and RPR ($p>0.05$). The analysis of the ROC curve for NLR revealed that it has a significant predictive ability for AP ($R2 = 0.842$; $p<0.001$). If the cut off value for the NLR parameter is set to 4,1030, then the sensitivity is 71.4% and the specificity is 100%. C-reactive protein (CRP) and glucose had a statistically significant positive correlation with NLR ($p=0.001$,

p=0.043). Ranson was close to being statistically significant (p = 0.051). The conclusion of the study was that NLR may serve as an early indicator of AP and as a predictor of disease severity.

Yang ZW, Mengb XX, Xub P (2015)[48] examined that Severe acute pancreatitis (SAP) is an acute gastrointestinal condition with a high systemic inflammatory response that rapidly progresses from local pancreatic injury to multiple organ failure. For many decades, it was believed that the contributions of neutrophils to the pathogenesis of SAP were the inflammatory chemokine and cytokine cascades. In this review, we are mostly concerned with the recently identified characteristics of neutrophils in SAP processes. Emerging research suggests that treatment strategies targeting neutrophils dramatically reduce tissue damage and prevent pancreatitis. Second, trypsin activation stimulates the first migration of neutrophils into the pancreas, and neutrophil infiltration in turn stimulates trypsin synthesis. In the absence of pathogens, neutrophils have the unique capacity to release neutrophil extracellular traps. In pancreatic acinar cells, the coordinated interaction between neutrophils and trypsinogen is essential for regulating the beginning, development, and resolution of inflammation. Neutrophil function in the progression of pancreatitis has led to the development of a novel technique to eliminate noxious chemicals and restore tissue homeostasis.

Bilgic Y et al (2016)[49] analyzed the link between platelet indices and the severity of acute pancreatitis (AP) and if there is any correlation. In the Department of Gastroenterology, Inonu University Faculty of Medicine, 47 patients with AP were classified into two groups: those with severe AP (n=15) and those with mild AP (n=32). The scores of these patients on days 0, 3, and at the start of remission according to prognostic scoring methods (Ranson, APACHE II, and BISAP criteria), CRP, sedimentation, and platelet indices (platelet count, MPV, PDW, and PCT) were recorded. Using the Mann-Whitney U test, the groups were compared. The

associations between platelet indices and other prognostic variables were evaluated using Spearman's Rho test. Platelets (P<0.02), PDW (P<0.001), AST (P<0.001), ALT (P<0.001), Total bilirubin (P<0.007), LDH (P<0.001), CRP (P<0.001), sedimentation (P<0.001), APACHE-II (P<0.001), and Ranson (P<0.02) characteristics differed significantly across groups. Platelet indices demonstrated a substantial positive or negative connection with other prognostic scoring systems, CRP and sedimentation, in patients with mild AP. Platelet indices in severe AP cases did not exhibit a strong association with other prognostic scoring systems, including CRP and sedimentation. Platelet derivatives can be employed as a promising predictive biomarker for the progression of moderate AP, the study concluded. In situations of severe AP, however, the applicability of platelet indices must be substantiated by additional research.

Obied ASH, Humeida AAK (2017)[50] Sudanese individuals with acute pancreatitis were evaluated for Prothrombin Time, Activated Partial Thromboplastic Time, Platelet Count, and Serum Amylase. In a case control study Sixty samples from Sudanese patients with acute pancreatitis were analysed for Prothrombin Time (PT), Activated Partial Thromboplastic Time (APTT), Platelet count, and serum amylase. Thirty samples were obtained from patients (case group) and thirty samples were obtained from healthy individuals (control group). The ethics commission of the ministry of health approved this study, and informed consent was obtained from each participant prior to sample collection. Patients at IbnSen hospital were surveyed using a standardized questionnaire approach to acquire data. The PT and APTT were performed using a semi-automated coagulometer (COATRON-MI) and a Sysmex Kx-21N plate reader. The data were examined with SPSS21. In this study, the mean SD of age PT, PTT, and Platelets in the case group were 59.33 (±7.36), 31.06 (± 6.29), and 57.33 (±9.20) seconds, respectively. In contrast, the mean SD of PT, PTT, and Platelets in the

control group were 13.33 (±1.15), 34.94 (±2.59), and 287.43 (± 47.59)x109 /L, respectively. (P=0.00) The difference was statistically significant. The conclusion of the study showed that PT and APTT were lengthened in patients with acute pancreatitis compared to normal participants, although the platelet count was normal in all patients.

Kaplan M et al (2018)[51] Examined the predictive significance of the platelet-lymphocyte ratio (PLR) and neutrophil-lymphocyte ratio (NLR) combination in patients with acute pancreatitis and its association with death. In this retrospective analysis, 142 patients with acute pancreatitis were included. Patients' Ranson, Atlanta, and BISAP 0h, 24h, and 48h scores were determined by checking their patient records. The patients were split into three groups based on their PLR and NLR levels: low-risk, medium-risk, and high-risk. The number of patients with acute pancreatitis complications such as necrotizing pancreatitis, acute renal failure, sepsis, and cholangitis was considerably higher in the high-risk group than in other groups, according to the findings of the present study. The high-risk group had a mortality rate of 90%, the medium-risk group 16%, and the low-risk group 1.9%. As comparison to other groups, the proportion of patients with a Ranson score of 5 and 6, a severe Atlanta score, a BISAP 0h score of 3 and 4, and BISAP 24h and 48h scores of 4 and 5 was greater in the high-risk group. In a stepwise regression model, the PLR-NLR combination, Atlanta and Ranson scores, and C-reactive protein level were identified as independent risk variables predicting mortality. In terms of predicting acute pancreatitis prognosis, the PLR-NLR combination exhibited the highest area under the curve and comparable diagnostic discrimination to other scoring methods. The study concluded that the PLR-NLR combination had the same prognostic significance as other grading systems used to predict the prognosis of acute pancreatitis.

Chopra R, Chauhan A, Thakur CS, Dogra A, Thakur S (2019)[52] Assessed the severity of acute pancreatitis using blood platelet levels as a

measure. After obtaining informed consent, 37 patients with acute pancreatitis admitted to the Department of Surgery at IGMC Shimla were included in the study. Individuals with acute pancreatitis were investigated and diagnosed utilizing blood tests (serum amylase) and imaging modalities (ultrasound and contrast enhanced CT scan). On day 1 and day 5 of admission, individuals with mild and severe pancreatitis had their blood platelet count compared. The results were then statistically analysed. A platelet count of 150 m/ mm3 was statistically significant on both day 1 (p=0.008) and day 5 (p=0.001) of admission between patients with moderate and severe pancreatitis, according to the findings of this study. The authors found that platelet count can serve as a solid, cost-effective prognostic indicator for determining the severity of pancreatitis.

Yalçın MS, Yalaki S (2019)[53] The objective of this study was to determine if there was a difference in predicting AP-related mortality between current scores and indirect markers of systemic inflammation, namely red cell distribution width (RDW), mean platelet volume (MPV), platelet lymphocyte ratio (PLR), and neutrophil-lymphocyte ratio (NLR) at the 48th hour. In a retrospective analysis, the 48-hour acute phase reactant levels of 667 AP patients admitted to the Gastroenterology departments of two hospitals were included. CRP0 represents the CRP value at the time of presentation, while CRP, RDW, MPV, NLR, and PLR represent values at the 48-hour mark. Ranson score, Atlanta score, NLR, and PLR values were determined for each patient. The patients were categorized as survivors (n = 641, or 96.1%) and non-survivors (n = 26, or 3.9%). NLR and PLR were found to be considerably greater in the non-survivor group (P0.001 and P=0.02, respectively) according to the findings of the present investigation. Both NLR and PLR had a weak and favourable relationship with mortality. The study found no correlation between MPV and mortality, although RDW was weakly and positively associated. Nonetheless, the scientists discovered that NLR and PLR values obtained 48 hours after AP administration are accurate indicators for predicting death. As appropriate,

these readily available and inexpensive diagnostics may be performed to monitor these patients more closely.

Reddy CS, Karimaddela K, Theja P, Prakash GV, Kumar PM (2019)[1] The ratio of platelets to lymphocytes (PLR) is a relatively novel inflammatory marker that can predict the severity of a variety of disorders. In this study, the authors assessed the predictive usefulness of PLR in acute pancreatitis (AP). This prospective study was conducted on 256 patients diagnosed with acute pancreatitis and admitted to S.V.R.R.G.G. Hospital between April 2017 and September 2018. Patients with Acute Pancreatitis were categorized into tertiles based on PLR values upon admission, with a PLR of less than 150 as the first tertile, 150-300 as the second tertile, and more than 300 as the third tertile. Admission to the intensive care unit (ICU), hospital length of stay (LOS), and mortality were evaluated as outcomes. Compared to those in the first tertile (PLR150), patients in the third tertile (PLR>300) had considerably more ICU admissions, 59 (71%), longer average Length of Hospital Stay (LOS) of survivors, 18+/-5 days, and greater death, 24 (28.9%). This study indicated that PLR is a simple, cost-effective prognostic indicator that can be used to predict the outcome of acute pancreatitis. The authors of this study determined that a high PLR score is related with a very poor prognosis and poor result or mortality in patients with acute pancreatitis.

Gokden Y, Kutlu O (2020)[33] Examined serum mean platelet volume (MPV) and platelet/lymphocyte ratio (PLR) levels in individuals with acute pancreatitis (AP), assessing whether MPV and PLR accurately predict AP disease severity at an early stage. Included in the study were 140 patients diagnosed with AP and 49 healthy controls (HC). Through hospital registration systems, Ranson criteria, revised Atlanta criteria, and Balthazar scores of AP patients were identified, and mild-to-severe AP patients were segregated. Patients' MPV and PLR levels upon admission were evaluated. MPV levels were not significantly different between the

AP and HC groups (p=0.998), whereas PLR levels were significantly greater in the AP group than in the HC group (p<0.001). Mild-severe AP patients did not differ in terms of MPV according to Ranson criteria, revised Atlanta criteria, and Balthazar grading (p:0.355; p:0.276; p:0.634, respectively). According to new Atlanta criteria, PLR was shown to be greater in the severe AP group (p:0.023). However, there was no correlation found between PLR and Ranson and Balthazar scores (p:0.311, p:0.415, respectively). By categorizing AP patients as biliary or non-biliary based on their etiology, it was determined that non-biliary AP patients had a lower MPV (p:0.034). Regarding PLR, there was no difference between groups (p:0.0772). The authors found that MPV is unsuitable for early differentiation of mild-severe AP patients. PLR may be performed as a supportive test to estimate the severity of AP in patients admitted for treatment.

Elfatth AMA, Mekky AESM, Othman AM, Mohammed SM, Mokhtar MAE (2022)[54] analyzed the predictive usefulness of neutrophil-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) for severe acute pancreatitis. Between 2018 and 2020, a prospective cohort study was done at the Department of Tropical Medicine and Gastroenterology at Assiut University Hospital. One hundred AP patients were enrolled in the trial. These patients' demographic, clinical, laboratory, and outcome data were obtained. Both NLR and PLR were computed. According to the updated Atlanta Criteria, 19 (19%) of the enrolled patients had SAP and 81 (81%) had mild AP. Individuals with SAP had significantly increased serum creatinine, NLR, and PLR levels. Additionally, mortality and admission to an intensive care unit were substantially more common among individuals with SAP. PLR at cutoff >187.04 showed 87% overall accuracy with an AUC of 0.850, whereas NLR at cutoff >2.43 had 100% overall accuracy with an AUC of 1. The authors found that the prognosis for patients with SAP was poor. Using NLR and PLR can aid in identifying patients with

NLR and PLR. Recommendations are made for further research to corroborate these results.

MATERIAL & METHODS

SOURCE OF DATA

The present prospective study was conducted, after approval from institutional ethics committee (IEC). Written, informed consent was obtained from all patients.

METHOD OF COLLECTION OF DATA

Sample size calculation:

It is assumed that the prevalence rate of acute pancreatitis in India is 7.9 % [55] based on previous data.

Assumptions:

Confidence Level = 95%

Precision (d) = ± 5%

For estimation of sample size, the following formula has been used

$n = (Z2\alpha \times P \times (1-P))/d2$

Where;

$Z\alpha$ = Value of standard normal variate corresponding to α level of significance

P = Likely value of parameter

Q = 1 – P

d = Margin of errors which is a measure of precision

With these assumptions the sample size works out as 112.

Inclusion Criterion: Patients presenting to the hospital with diagnosis of acute pancreatitis.

Exclusion Criterion: Patients were excluded from this study if they had any of the following:

1. Pancreatitis developed as a result of trauma.
2. Patients with age less than 18 years of age.
3. Patients with diagnosed case of hematological disorders, cancers and patients on chemotherapy.
4. Patients not giving consent to participate in study.

This prospective study included patients presenting with acute pancreatitis in Dayanand medical college and hospital between 1st March 2021 to 28th Feb 2022. At least two of the following three symptoms will be sought-out criteria for Acute Pancreatitis diagnosis: acute condition presenting with abdominal pain, a threefold or greater rise in serum levels of pancreatic enzymes amylase or lipase and characteristic findings of pancreatic inflammation on imaging methods (including abdominal sonography and computed tomography). Patients with pancreatitis developed as result of trauma, patients with age less than 18 years of age, patients with haematological disorders, cancers and patients on chemotherapy, patients not giving consent to participate in study were excluded from study. Demographic characteristics, vital signs and laboratory parameters (white blood count, neutrophil count, lymphocyte count, haemoglobin level, platelet count, Red cell distribution width, mean platelet volume,

haematocrit) will be assessed and will be recorded in all patients. Platelet to lymphocyte ratio was calculated as ratio of absolute platelet count to absolute lymphocyte count at the time of presentation to hospital and at 24^{th} and 48^{th} hour of presentation to hospital. Mean of PLR value at time of presentation, 24^{th} and 48^{th} hour of presentation to hospital was calculated. We divided the patients diagnosed with acute pancreatitis into 3 groups according to mean platelet to lymphocyte ratio of <150 as ist group, a platelet to lymphocyte ratio of 150-300 as 2^{nd} group and >300 as 3^{rd} group and length of hospital stay and need of ICU/HDU stay, DOR/death/DAMA/discharge, type of management, organ failure occurrence as defined by [shock(systolic blood pressure <90mm hg), pulmonary insufficiency (pao2 < 60 mm hg), renal failure(creatinine level > 2 mg/dl after fluid resuscitation), gastrointestinal bleeding (>500 ml/24 hr)] according to the ATLANTA criteria for acute pancreatitis and MODS(multiple organ dysfunction syndrome), complications of acute pancreatitis like peripancreatic fluid collections, pancreatic necrosis and infected necrosis, pancreatic pseudocyst, pancreatic ascites and pancreaticopleural fistulas, vascular complications, pancreatocutaneous fistula will be compared between the groups. The results were statistically analysed.

Duration of study: 1 year (from 1st March 2021 to 28th Feb 2022)

Outcome: Patients fulfilling the inclusion criteria were evaluated prospectively and final outcome was noted in view of:

1. Length of hospital stay
2. Need of ICU stay/ HDU stay
3. DOR/death/DAMA/discharge
4. Type of management
5. Organ failure occurrence/MODS
6. Complications of acute pancreatitis

Statistical Analysis

Data was described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations were done using (Statistical Package for the Social Science) SPSS 26 version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows. T test and ANOVA were used for comparing grouped data while chi square was used of analyzing categorial data. Receiver operating curve was used to find the cut off and. the value to test the predictive value. p value of <0.05 was considered to be significant.

OBSERVATION & RESULTS

Table 1: Age distribution of the study population

Age group	No. of cases	Percentage
≤ 30	18	13.3%
31-40	40	29.6%
41-50	39	28.9%
51-60	27	20.0%
> 60	11	8.1%
Total	**135**	**100.0%**

The maximum patients in the present study were present in age group 31-40 year (29.6%) followed by 41- 50 years (28.9%) ,51-60(8.1%) and >60 with 22%, and least were in 51-60 with 8% .

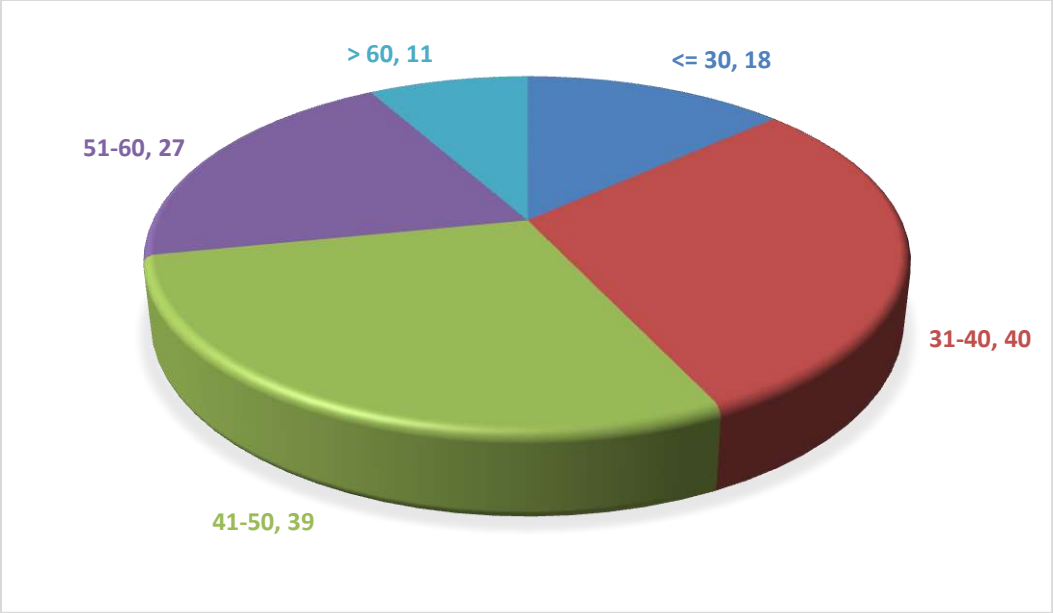

Figure 1: Age distribution of the study population

Table 2: Sex distribution of the study population

Sex	No. of cases	Percentage
Female	42	31.1
Male	93	68.9
Total	**135**	**100**

Males predominated the study. There were 68.9% males and 31.1% females in the study.

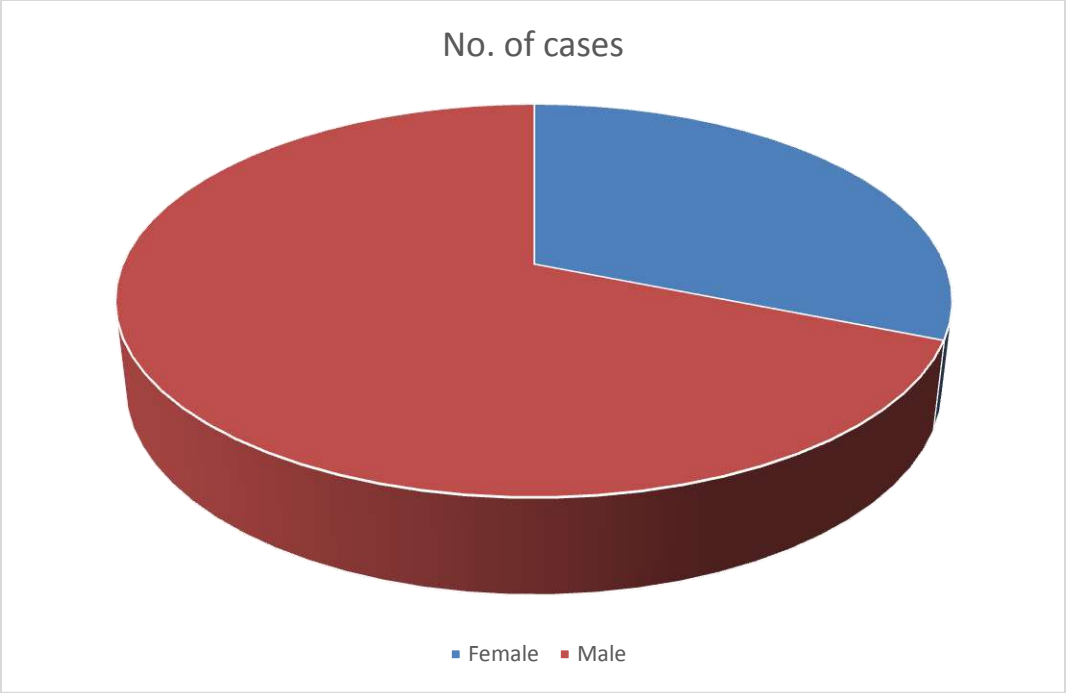

Figure 2: Sex distribution of the study population

Table 3: Distribution of patients according to etiology

Cause of pancreatitis	No. of cases	Percentage
Alcohol	50	37.0
Biliary	69	51.1
Hypercalcemia	8	5.9
Idiopathic	8	5.9
Total	**135**	**100**

In maximum patients gall stone was the major etiological factor with 51.1% (69) cases, followed by 37% (50) cases who had alcohol as an etiologic agent. In 5.9% (8) cases each hypercalcemia and idiopathic was the main reason for pancreatitis.

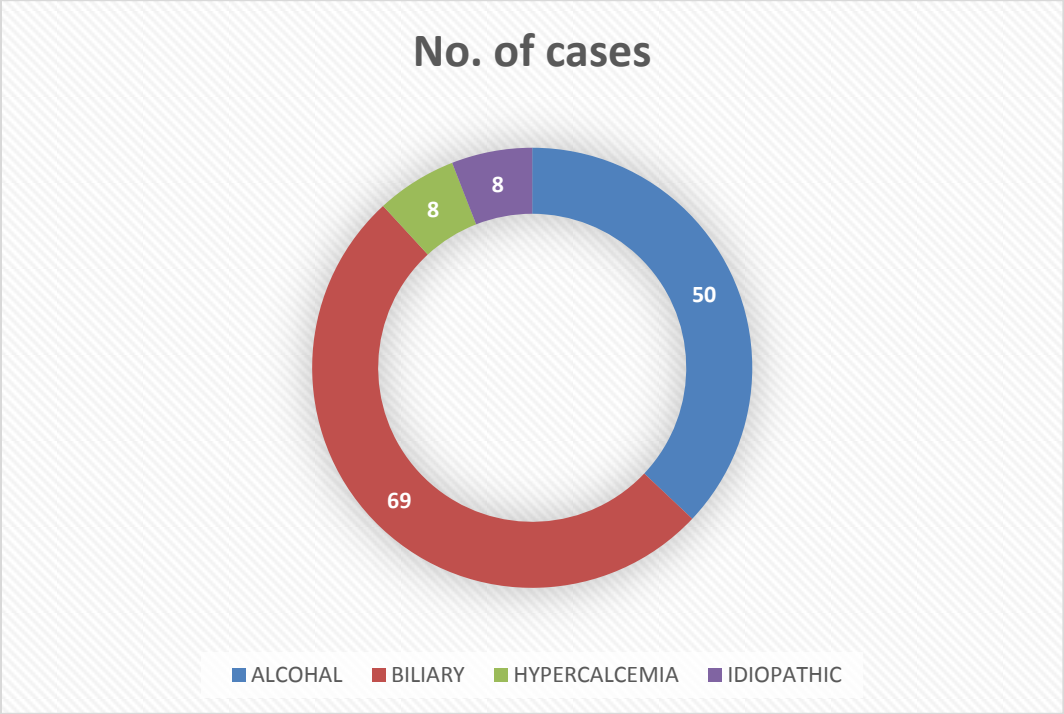

Figure 3: Distribution of cases according to etiology

Table 4: Distribution of patients according to platelets lymphocyte ratio

Platelets lymphocytes ratio	Baseline		24 hours		48 hours		Mean	
	No. of cases	%	No. of cases	%	No. of cases	%	No. of cases	%
≤150	51	37.8	42	31.1	64	47.4	49	36.3
151-300	70	51.9	77	57.0	66	48.9	76	56.3
>300	14	10.4	16	11.9	5	3.7	10	7.4
Total	**135**	**100**	**135**	**100**	**135**	**100**	**135**	**100**

At baseline 51(37.8%), at 24 hour 42(31.1%) and at 48 hours 64(47.4%) patients have PLR ≤150.

At baseline 70(51.9%), at 24 hour 77(57%) and at 48 hours 66(48.9%) patients have PLR 151-300.

At baseline 14(10.4%), at 24 hour 16(11.9%) and at 48 hours 5(3.7%) patients have PLR ≤150.

Comparing mean the mean PLR was ≤150 in 49(36.3%), it was 151-300 in 76(56.3%), >300 in 10(7.4%)

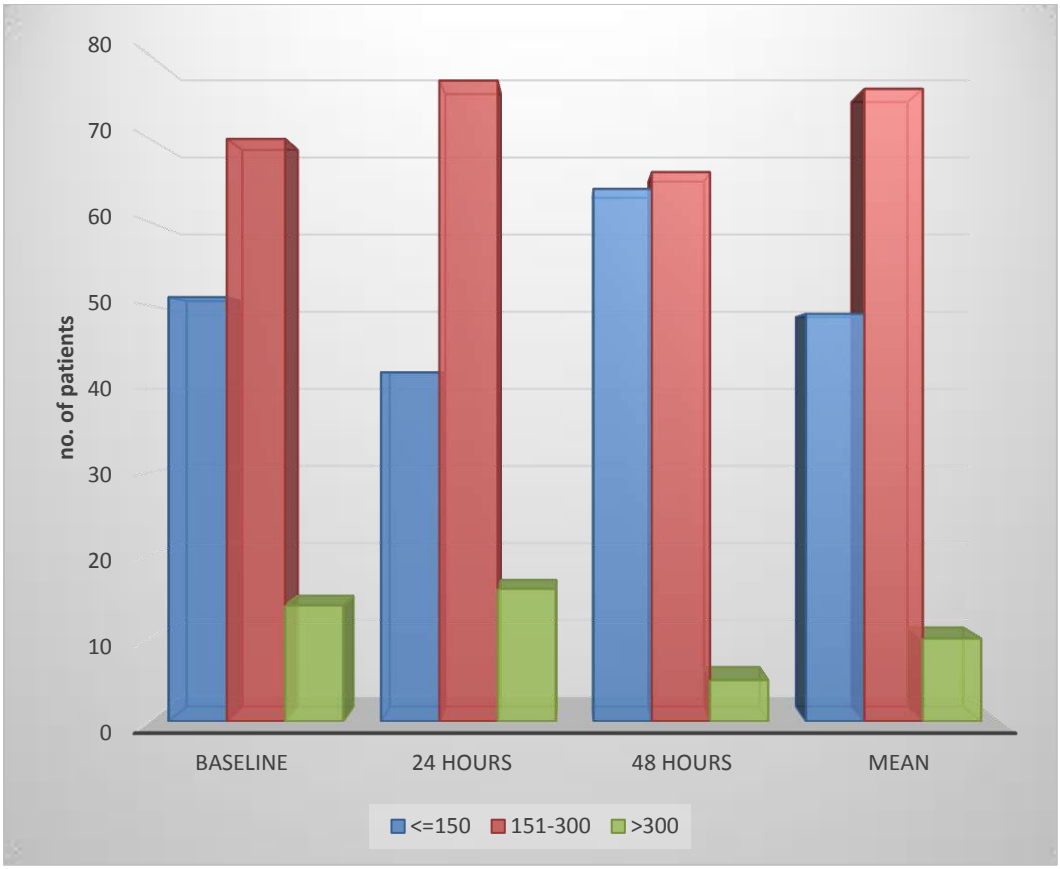

Figure 4: Distribution of patients according to platelets lymphocyte ratio

Table 5: Distribution of patients according to type of management

Type of management	No. of cases	Percentage
Conservative	117	86.7
Percutaneous catheter drainage	15	11.1
Surgical	3	2.2
Total	**135**	**100.0**

Majority of patients were managed conservatively 117(86.7%) while percutaneous catheter drainage was done in 15(11.1%). 3 (2.2%) were surgically managed.

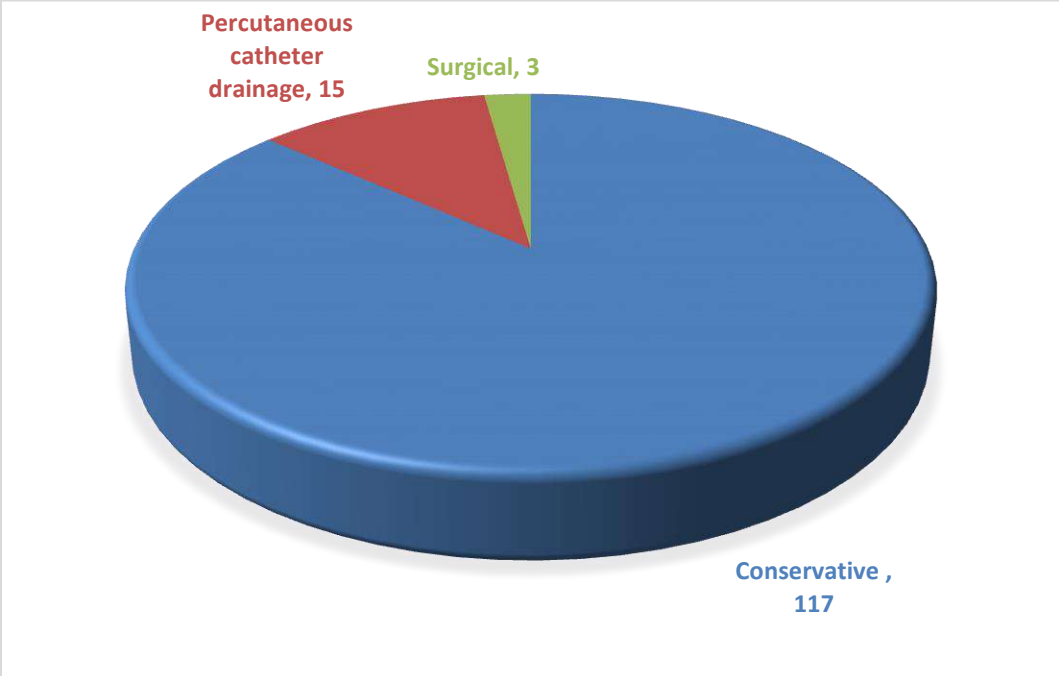

Figure 5: Distribution of patients according to type of management

Table 6: Distribution of patients according to condition of patients

Condition of patients	No. of cases	Percentage
Organ failure occurrence	34	25.2
Complications of acute pancreatitis	63	46.7
Need of ICU and HDU stay	45	33.3

Organ failure was seen in 34(25.2%) patients while complications due to acute pancreatitis were seen in 63(46.7%). 45(33.3%) patients needed ICU.

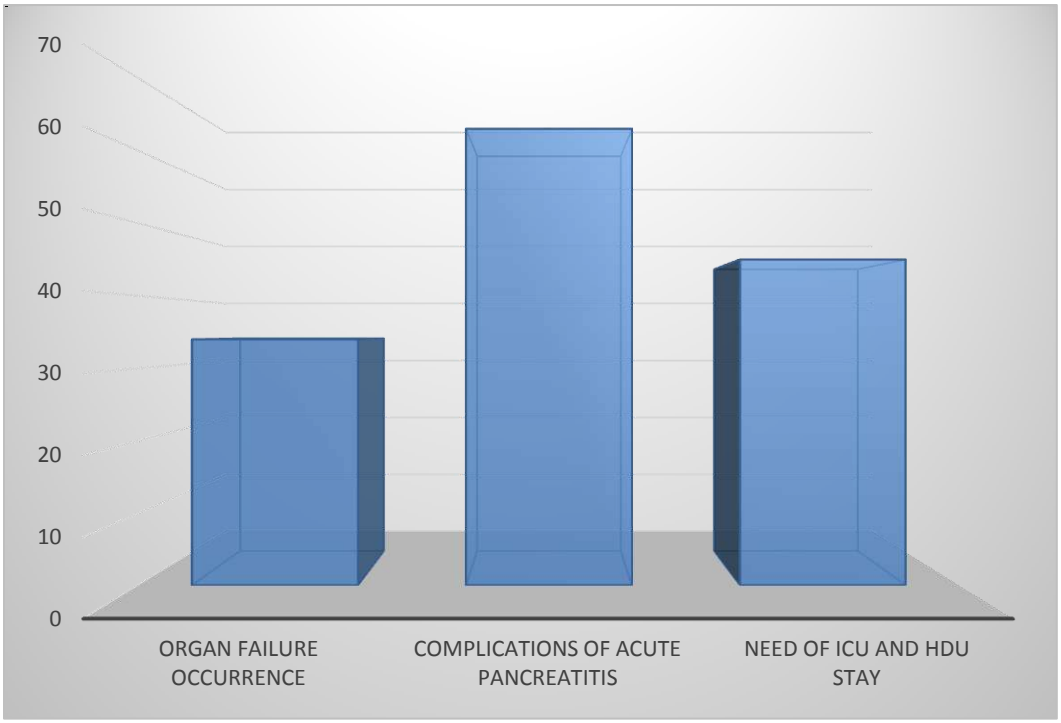

Figure 6: Distribution of patients according to condition of patients

Table 7: Distribution of patients according to outcome

DOR/Death/DAMA/Discharge	No. of cases	Percentage
DAMA	11	8.1
Discharge	110	81.5
DOR	14	10.4
Total	**135**	**100.0**

110(81.5%) patients were successfully treated and were discharged, 11% discharged against medical advice and 14(10.4%) were discharged on request

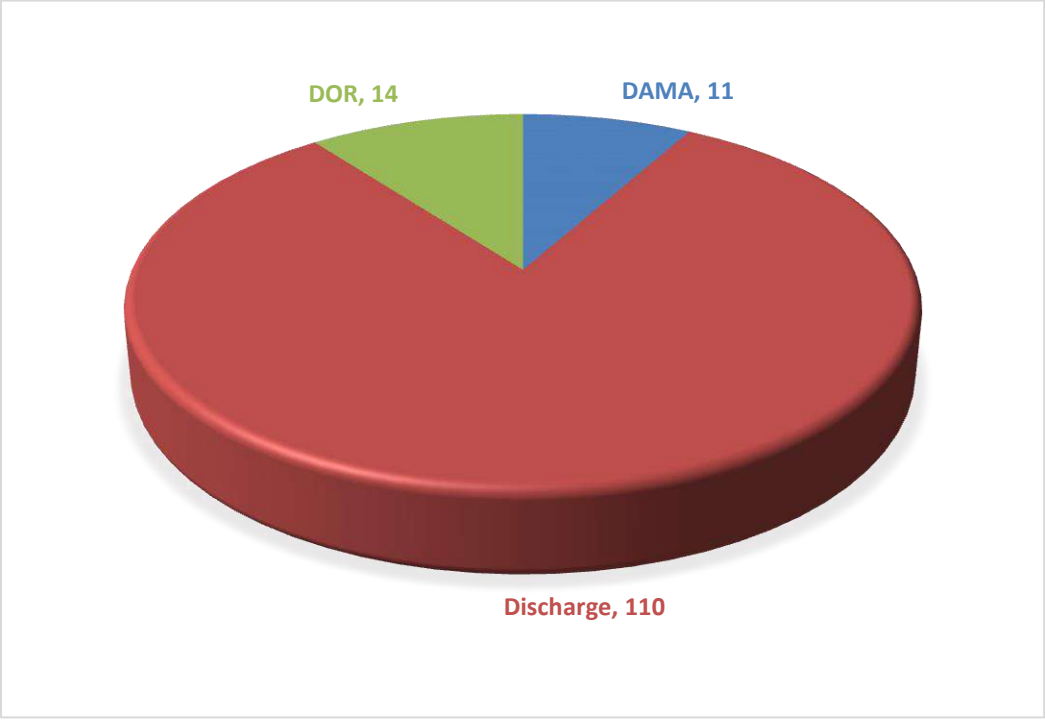

Figure 7: Distribution of patients according to outcome

Table 8: Mean values of grouped variables

	Min	Max	Mean	Std. Deviation
Age	18.00	88.00	43.83	13.66
Serum amylase	235.00	950.00	330.07	88.60
Serum lipase	250.00	1210.00	660.51	212.47
Serum calcium	4.10	9.50	8.54	0.87
Lymphocyte 0hr	450.00	3296.00	1472.79	501.17
Lymphocyte at 24hrs	410.00	3426.00	1469.16	512.13
Lymphocyteat4 8hrs	523.00	3776.00	1676.82	599.90
PLTs 0hr	93000.0	450000.0	256775.6	66456.3
PLTs 24hrs	92000.0	420000.0	260537.8	62688.3
PLTs 48hrs	94000.0	411200.0	263745.2	62034.7
PLR 0hr	67.84	422.86	190.77	72.66
PLR 24hrs	72.97	411.80	194.73	72.34
PLR 48hrs	66.26	556.41	174.16	69.70
Mean PLR	69.21	421.32	186.55	67.84
Length of hospital stay	3.00	47.00	11.08	7.04

Table 8 shows the descriptive value of various variables of the present study.

Table 9: Comparing lymphocytes at baseline, 24 hours and 48 hours

Lymphocytes *per µl*	Mean	SD	t	*p* value	Mean Difference	SD difference
Baseline	1472.79	501.17				
24hrs	1469.16	512.13	0.339	0.735	3.622	124.267
4 8hrs	1676.82	599.90	-7.534	0.001	-204.037	314.655

The mean lymphocyte per *µl* at baseline was 1472±501.70, at 24 hours was 1469.16±512.13 and at 48 hours was 1676.82±599.90. lymphocytes were significantly more at 48 hours then baseline and 24 hours. However, at 24 hours no significant difference was seen between the two.

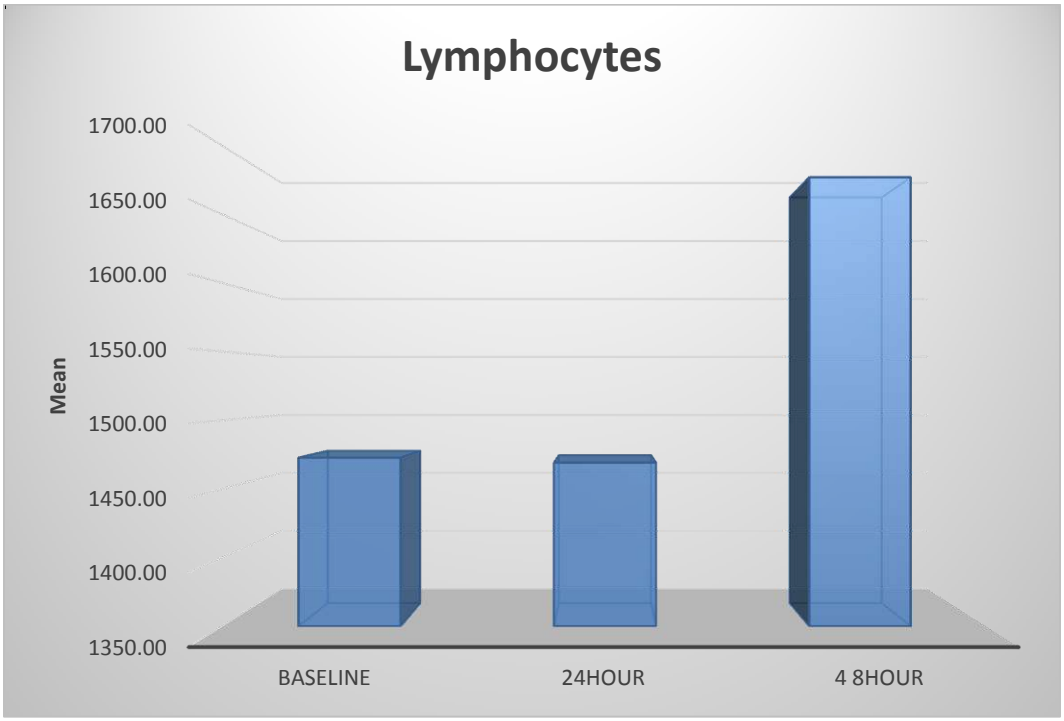

Figure 8: Comparing lymphocytes at baseline, 24 hours and 48 hours

Table 10: Comparing platelets at baseline, 24 hours and 48 hours

Platelets/*µl*	Mean	SD	t	p-value	Mean Difference	SD difference
Baseline	256775.56	66456.27				
24 hrs	260537.78	62688.33	-2.748	0.007	-3762.222	15909.154
48 hrs	263745.19	62034.68	-4.158	0.001	-6969.630	19474.263

The mean platelets per µl at baseline was 256775.56± 66456.27, at 24 hours was 260537.78 ± 62688.33 and at 48 hours was 263745.19±62034.68. Platelets were significantly more at 24 hours and 48 hours then baseline.

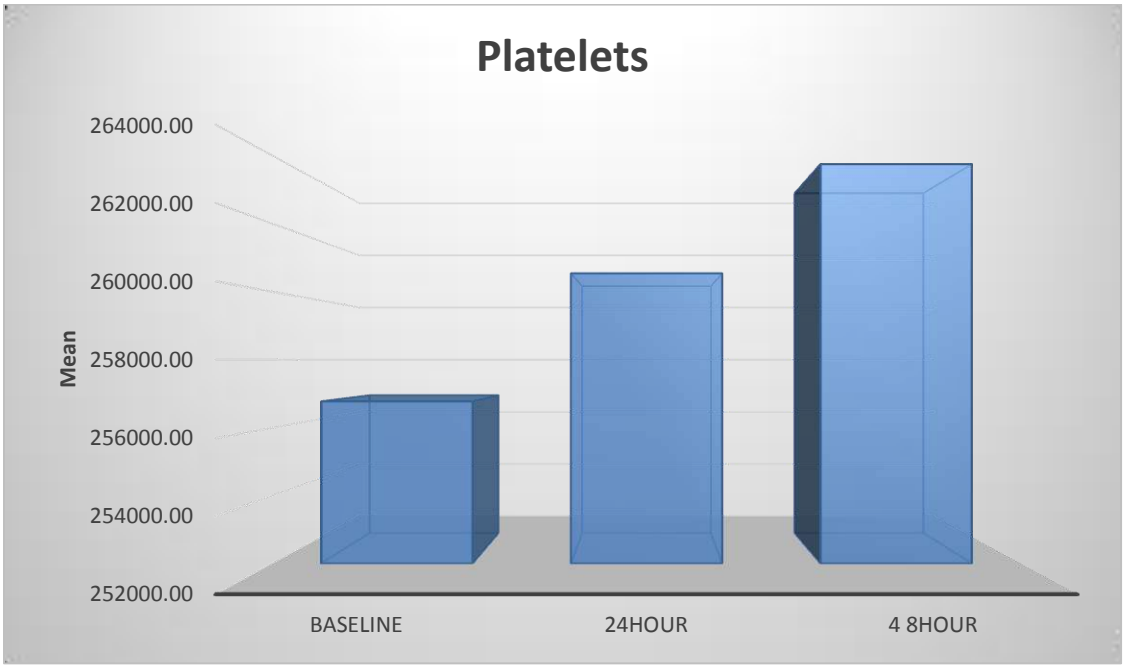

Figure 9: Comparing platelets at baseline, 24 hours and 48 hour

Table 11: Comparing platelets lymphocyte ratio at baseline, 24 hours and 48 hours

PLR	Mean	Std. Deviation	t	p-value	Mean Difference	SD difference
Baseline	190.77	72.66				
24 hrs	194.73	72.34	-2.218	0.028	-3.956	20.727
48 hrs	174.16	69.70	3.891	0.001	16.611	49.603

The mean PLR at baseline was 190.77± 72.66, at 24 hours was 194.73±72.34 and at 48 hours was 174.16 ± 69.70. PLR were significantly more at 24 hours and 48 hours then baseline.

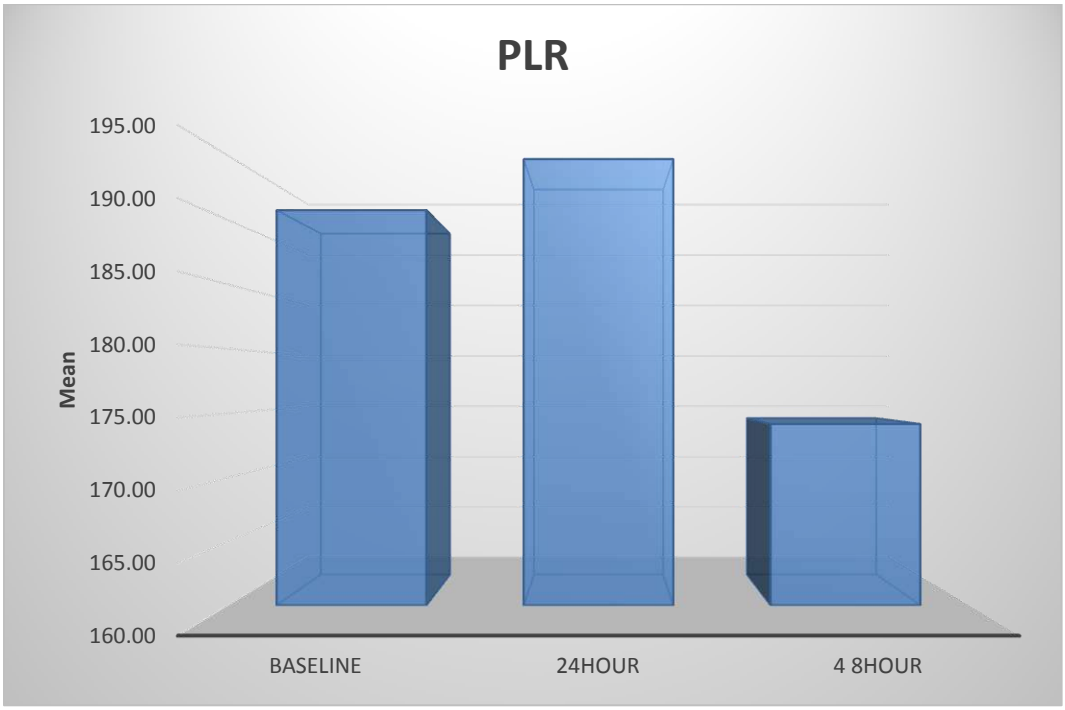

Figure 10: Comparing platelets lymphocyte ratio at baseline, 24 hours and 48 hours

Table 12: Association of different grades of PLR ratio with that of different variables

Variable	Subgroups	Baseline						Total	Chi-square value	p-value
		≤150		151-300		>300				
		N	%	N	%	N	%			
Age group	< 30	7	14	8	11	3	21	18	9.384	0.311
	31-40	18	35	20	29	2	14	40		
	41-50	9	18	23	33	7	50	39		
	51-60	13	25	13	19	1	7	27		
	> 60	4	8	6	9	1	7	11		
Sex	Female	13	25	24	34	5	36	42	1.219	0.544
	Male	38	75	46	66	9	64	93		
Type of management	Conservative	47	92	59	84	11	79	117	12.153	0.016
	Percutaneous catheter drainage	4	8	10	14	1	7	15		
	Surgical	0	0	1	1	2	14	3		
Organ failure occurrence	Absent	46	90	50	71	5	36	101	18.189	0.016
	Present	5	10	20	29	9	64	34		
Complications of acute pancreatitis	Absent	31	61	36	51	5	36	72	2.986	0.225
	Present	20	39	34	49	9	64	63		

Need of ICU and HDU stay	Absent	38	75	45	64	7	50	90	3.34	0.188
	Present	13	25	25	36	7	50	45		
DOR death DAMA discharge	DAMA	6	12	2	3	3	21	11	7.106	0.130
	Discharge	39	76	61	87	10	71	110		
	DOR	6	12	7	10	1	7	14		
Total		**51**	**100**	**70**	**100**	**14**	**100**	**135**		

We observed that at baseline people with PLR ≤150 were mostly treated conservatively, while between 151-300 84% were treated conservatively and >300 79% were treated conservatively. While 14% of patients with PLR>300 had surgery. This association was significant. Similarly, complications were mostly seen in patients who had PLR>300(64%) vs PLR 151-300(49%) and ≤150(39%).

Similarly, organ failure was mostly seen in patients who had PLR>300(64%) vs PLR 151-300(29%) and ≤150(10%). Need for ICU were mostly seen in patients who had PLR >300(50%) vs PLR 151-300(36%) and ≤150(25%). This association was not significant.

DOR was more in patients with PLR≤150, DAMA was more in patients with PLR 151-300, patients who had PLR ≤150. this association was not significant.

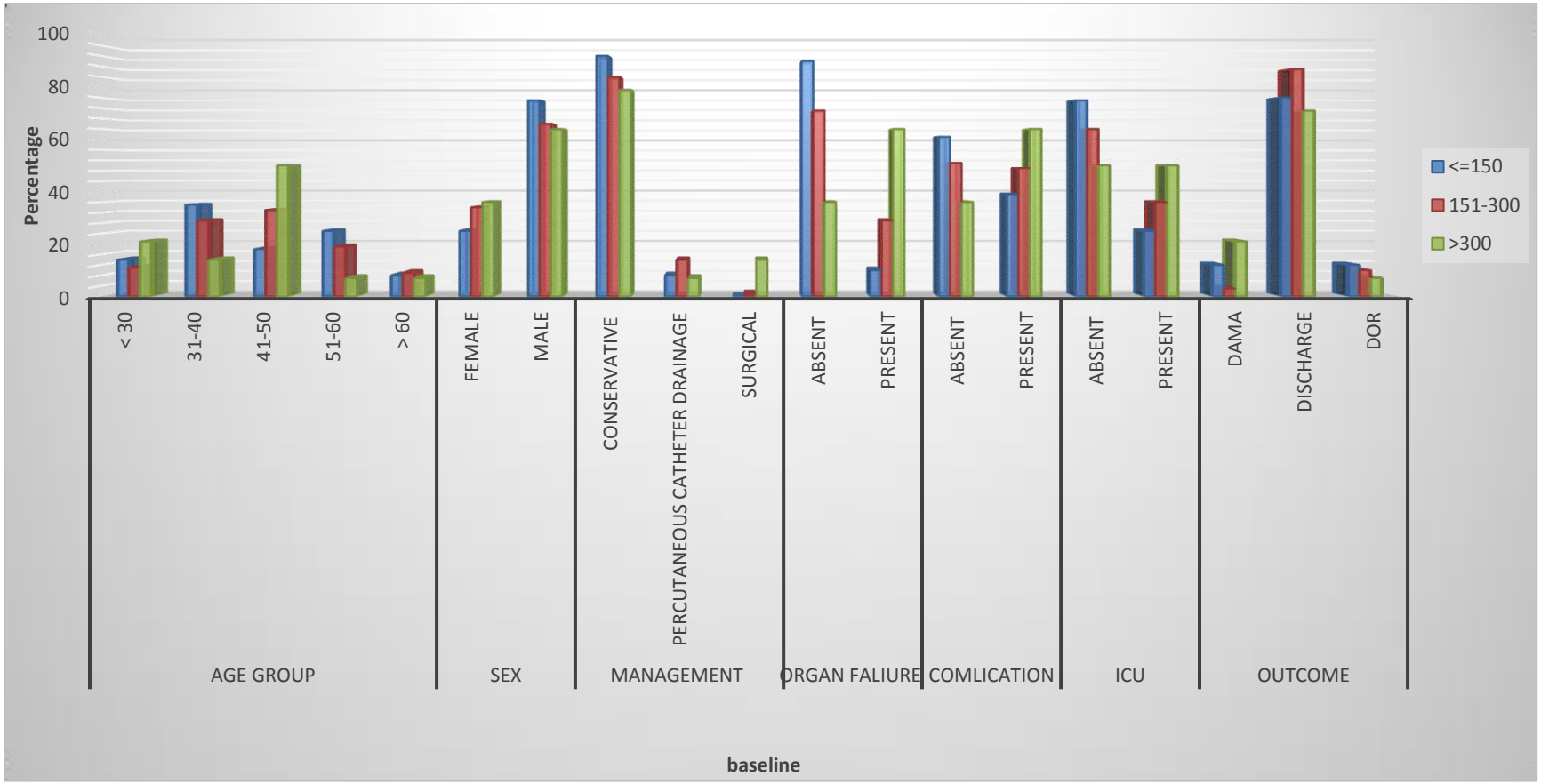

Figure 11: Association of different grades of PLR ratio with that of different variables

Table 13: Association of different grades of PLR ratio with that of different variables at 24 hours

		PLR at 24 hours						Total	Chi-square value	p-value
		≤150		151-300		>300				
		n	%	n	%	n	%			
Age group	< 30	4	10	11	14	3	19	18	7.743	0.459
	31-40	16	38	22	29	2	13	40		
	41-50	9	21	22	29	8	50	39		
	51-60	10	24	15	19	2	13	27		
	> 60	3	7	7	9	1	6	11		
Sex	Female	10	24	27	35	5	31	42	1.607	0.448
	Male	32	76	50	65	11	69	93		
Type of management	Conservative	38	90	68	88	11	69	117	23.146	0.001
	Percutaneous catheter drainage	4	10	9	12	2	13	15		
	Surgical	0	0	0	0	3	19	3		
Organ failure	Absent	39	93	57	74	5	31	101	23.398	0.001

occurrence	Present	3	7	20	26	11	69	34		
Complications of acute pancreatitis	Absent	28	67	39	51	5	31	72	6.358	0.042
	Present	14	33	38	49	11	69	63		
Need of ICU and HDU stay	Absent	33	79	50	65	7	44	90	6.564	0.038
	Present	9	21	27	35	9	56	45		
DOR death DAMA discharge	DAMA	6	14	1	1	4	25	11	13.343	0.010
	Discharge	32	76	68	88	10	63	110		
	DOR	4	10	8	10	2	13	14		
Total		**42**	**100**	**77**	**100**	**16**	**100**	**135**		

We observed that at 24 hour patients with PLR ≤150(90%) and 151-300 (88%)were mostly treated conservatively , >300, 69% were treated conservatively. While 19% of patients with PLR >300 had surgery. This association was significant. Similarly, complications were mostly seen in patients who had PLR >300(64%) vs PLR 151-300(49%) and ≤150(39%).

Similarly, organ failure was mostly seen in patients who had PLR >300(69%) vs PLR 151-300(26%) and ≤150(7%). Need for ICU were mostly seen in patients who had PLR >300(56%) vs PLR 151-300(35%) and ≤150(21%). This association was significant.

DOR was more in patients with PLR ≤150 (10%) and PLR 151-300(10%), DAMA was more in patients with PLR >300(25%), discharge was more in patients who had PLR 150-300(76%), ≤150(88%) and>300(63%). this association was significant.

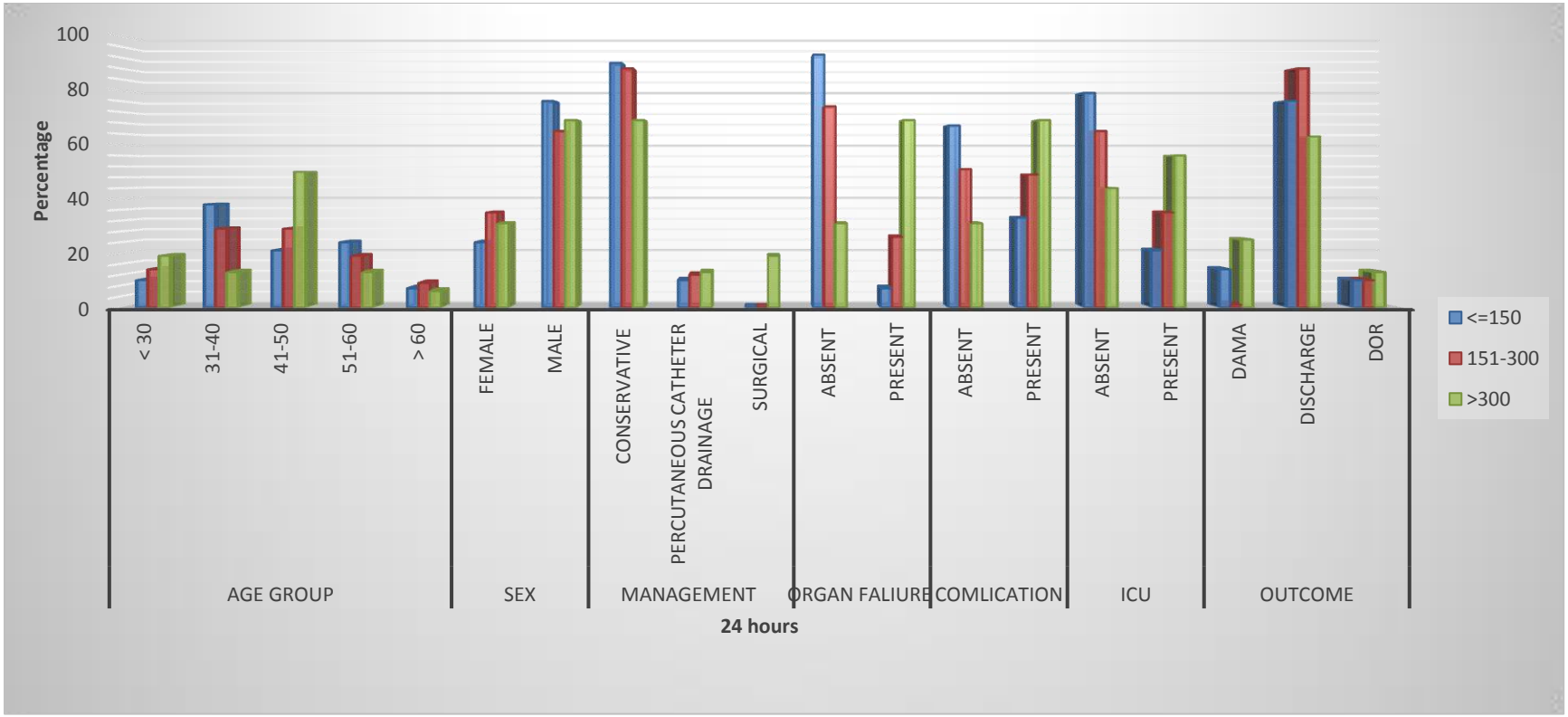

Figure 12: Association of different grades of PLR ratio with that of different variables at 24 hours

Table 14: Association of different grades of PLR ratio with that of different variables at 48 hours

		PLR at 48 hours						n	Total	Chi-square value
		≤150		151-300		>300				
		n	%	n	%				%	n
Age group	< 30	6	9	12	18	0	0	18	9.005	0.342
	31-40	23	36	16	24	1	20	40		
	41-50	16	25	20	30	3	60	39		
	51-60	15	23	12	18	0	0	27		
	> 60	4	6	6	9	1	20	11		
Sex	Female	17	27	23	35	2	40	42	1.232	0.54
	Male	47	73	43	65	3	60	93		
Type of management	Conservative	60	94	54	82	3	60	117	9.592	0.048
	Percutaneous catheter drainage	4	6	9	14	2	40	15		
	Surgical	0	0	3	5	0	0	3		
Organ failure occurrence	Absent	60	94	40	61	1	20	101	27.223	0.001
	Present	4	6	26	39	4	80	34		

Complications of acute pancreatitis	Absent	46	72	25	38	1	20	72	17.406	0.001
	Present	18	28	41	62	4	80	63		
Need of ICU and HDU stay	Absent	52	81	36	55	2	40	90	12.089	0.002
	present	12	19	30	45	3	60	45		
DOR death DAMA discharge	DAMA	6	9	2	3	3	60	11	20.554	0.001
	discharge	51	80	57	86	2	40	110		
	DOR	7	11	7	11	0	0	14		
Total		**64**	**100**	**66**	**100**	**5**	**100**	**135**		

We observed that at 48-hour patients with PLR ≤150(94%) and 151-300 (82%) were mostly treated conservatively, >300, 60% were treated conservatively. While 14% of patients with PLR >300 had surgery. This association was significant. Similarly, complications were mostly seen in patients who had PLR >300(80%) vs PLR 151-300(62%) and ≤150(28%).

Similarly, organ failure was mostly seen in patients who had PLR>300(80%) vs PLR 151-300(39%) and ≤150(6%). Need for ICU were mostly seen in patients who had PLR >300(60%) vs PLR 151-300(45%) and ≤150(19%). This association was significant.

DOR was more in patients with PLR ≤150 (11%) and PLR 151-300(11%), DAMA was more in patients with PLR >300(60%), discharge was more in patients who had PLR 150-300(86%), ≤150(80%) and>300(40%). this association was significant.

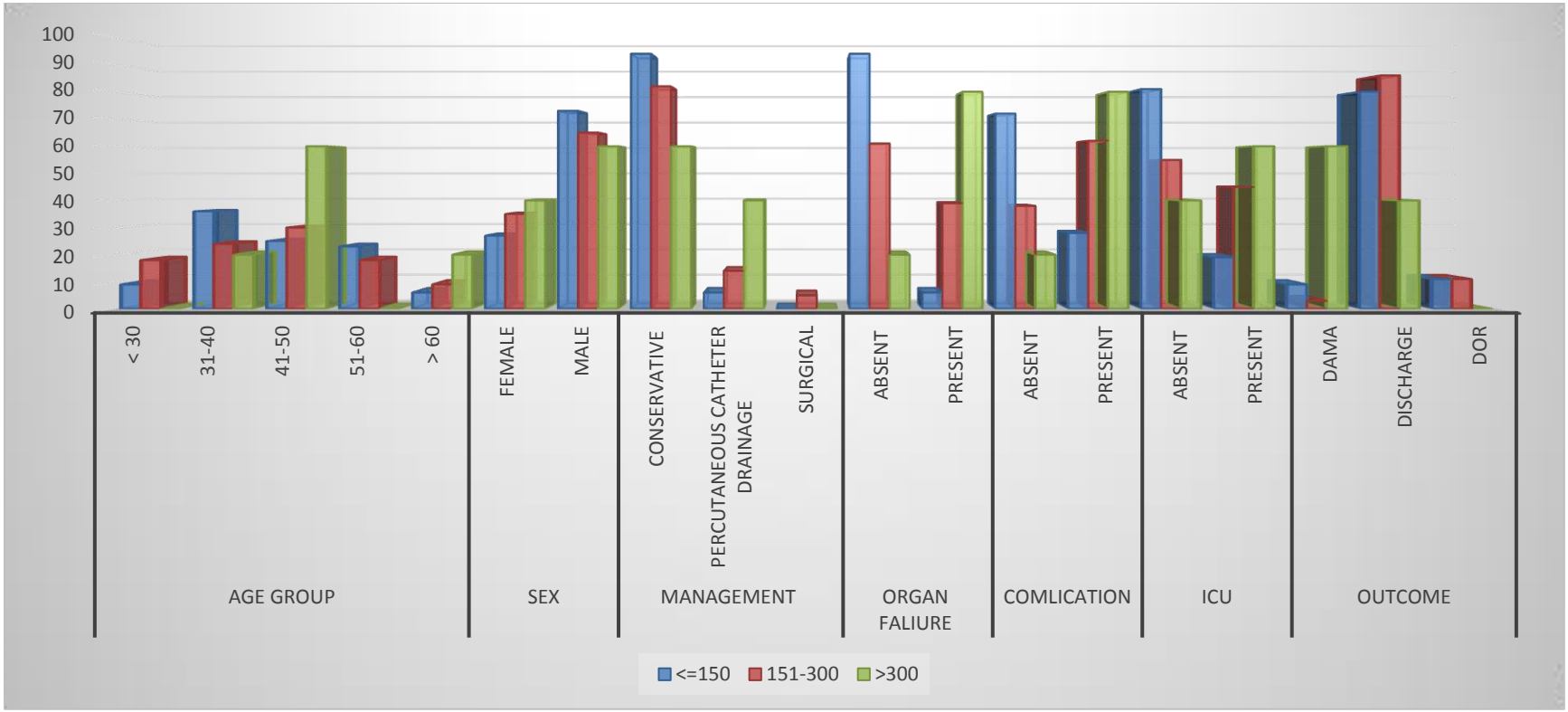

Figure 13: Association of different grades of PLR ratio with that of different variables at 48 hours

Table 15: Comparing various study parameters at baseline between different grades of PLR ratio

PLR at 0	≤150		151-300		>300		F	p-value
	Mean	SD	Mean	SD	Mean	SD		
Age	43.65	14.36	44.29	13.41	42.21	13.13	0.140	0.870
Serum amylase	324.47	63.52	330.21	105.33	349.71	77.30	0.442	0.643
Serum lipase	644.00	234.54	668.86	196.51	678.93	216.77	0.258	0.773
Serum calcium	8.50	0.70	8.55	0.88	8.59	1.31	0.071	0.931
Lymphocyte 0hr	1769.14	462.13	1385.77	407.84	828.29	228.31	31.288	0.001
Lymphocyte at 24 hrs	1776.12	484.60	1377.71	403.80	808.21	222.86	32.127	0.001
Lymphocyte at 48hour	1951.33	546.25	1592.11	571.06	1100.36	372.68	15.139	0.001
PLTs 0hr	222384.31	45398.79	275884.29	67698.33	286514.29	75577.89	13.140	0.001
PLTs 24 hrs	230515.69	45683.25	278145.71	63328.79	281864.29	74153.69	10.797	0.001

PLTs 48 hrs	234478.43	49476.45	281384.29	61581.37	282164.29	68723.03	10.403	0.001
Length of hospital stay	7.86	4.50	12.10	7.02	17.71	8.84	14.793	0.001

We observed that no significant difference was seen among serum amylase, lipase, calcium levels. However, lymphocyte and platelets at baseline, 24 hour, and 48 hours had significant difference among the three groups. The hospital stay was also significantly more in ones who have PLR >300(17.71 ± 8.84) then 151-300 (12.10 ± 7.02) and ≤150(7.86 ± 4.50).

Table 16: Comparing various study parameters at 24 hours between different grades of PLR ratio

PLR at 24hrs	≤150		151-300		>300		F	p-value
	Mean	SD	Mean	SD	Mean	SD		
Age	43.48	13.53	44.19	14.09	43.00	12.63	0.070	0.932
Serum amylase	326.36	63.86	326.56	101.52	356.69	76.87	0.817	0.444
Serum lipase	654.24	238.99	659.43	201.35	682.19	202.85	0.101	0.904
Serum calcium	8.48	0.76	8.54	0.84	8.64	1.23	0.188	0.829
Lymphocyte 0 hr	1823.10	492.47	1410.40	386.28	853.44	228.41	34.670	0.001
Lymphocyte at 24 hour	1840.48	510.87	1399.96	379.41	827.50	224.74	37.562	0.001
Lymphocyte at 48 hour	2021.14	569.85	1606.21	539.67	1112.81	393.53	18.265	0.001
PLTs 0hr	223457.14	49742.22	268549.35	66831.27	287575.00	72310.55	9.211	0.001

PLTs 24hrs	230054.76	50000.47	272145.45	61432.00	284693.75	72360.75	8.286	0.001
PLTS 48hrs	234561.90	53658.39	275035.06	59972.91	286018.75	68677.55	7.644	0.001
Length of hospital stay	7.29	4.47	11.27	5.18	20.13	11.16	26.744	0.001

We observed that no significant difference was seen among serum amylase, lipase, calcium levels. However, lymphocyte and platelets at, 24 hours had significant difference among the three groups. The hospital stay was also significantly more in ones who have PLR >300(20.13±11.16) then 151-300 (11.27± 5.18) and ≤150(7.29±4.47)

Table 17: Comparing various study parameters at 48 hours between different grades of PLR ratio

PLR at 48 hrs	≤150		151-300		>300		F	p-value
	Mean	SD	Mean	SD	Mean	SD		
Age	43.84	13.07	43.38	14.35	49.60	13.07	0.478	0.621
Serum amylase	313.02	62.41	343.15	105.67	375.60	100.58	2.627	0.076
Serum lipase	621.59	222.42	702.33	196.43	606.60	222.51	2.572	0.080
Serum calcium	8.47	0.85	8.65	0.72	7.90	2.12	2.092	0.128
Lymphocyte 0hr	1713.47	521.88	1274.92	372.30	1003.80	156.47	18.572	0.001
Lymphocyte at 24hrs	1741.75	519.84	1245.91	360.61	927.00	196.07	24.501	0.001

Lymphocyte at4 8hrs	2058.67	574.39	1368.08	356.51	864.60	252.91	42.619	0.001
PLTs 0hr	237625.00	66388.48	268707.58	57982.57	344400.00	78017.95	9.035	0.001
PLTs 24hrs	244096.88	60921.78	270657.58	58218.14	337400.00	68682.60	7.479	0.001
PLTS 48hrs	248262.50	62323.52	273512.12	56967.70	333000.00	60004.17	6.405	0.002
Length of hospital stay	7.77	4.25	13.29	6.67	24.40	13.24	26.654	0.001

We observed that no significant difference was seen among serum amylase, lipase, calcium levels. However, lymphocyte and platelets at, 48 hours had significant difference among the three groups. The hospital stay was also significantly more in ones who have PLR >300(24.40±13.24) then 151-300 (13.29 ± 6.67) and ≤150(7.77±4.25).

Table 18: Comparing mean PLR ratio

PLR at Mean	≤150		151-300		>300		F	p-value
	Mean	SD	Mean	SD	Mean	SD		
Age	43.71	14.27	43.64	13.62	45.80	11.95	0.111	0.895
Serum amylase	325.92	64.08	327.75	102.38	368.00	76.72	0.996	0.372
Serum lipase	656.65	231.32	662.79	199.14	662.10	236.99	0.013	0.988
Serum calcium	8.52	0.72	8.55	0.85	8.49	1.55	0.040	0.961
Lymphocyte 0hr	1777.27	469.83	1365.14	408.46	798.90	254.80	27.758	0.001
Lymphocyte at 24hrs	1785.96	492.03	1357.12	403.14	768.40	247.97	29.210	0.001
Lymphocyte at 4 8hrs	1989.14	554.99	1568.76	545.79	967.70	211.49	18.810	0.001

PLTs 0hr	223020.41	46391.20	274725.00	66462.34	285760.00	87234.28	11.639	0.001
PLTs 24 hr	230583.67	46578.37	277185.53	62096.00	280790.00	84416.49	9.974	0.001
PLTs 48 hr	233873.47	50322.11	280636.84	60407.73	281740.00	75859.87	10.135	0.001
Length of hospital stay	7.24	4.23	11.95	5.52	23.30	11.68	34.262	0.001

We observed that no significant difference was seen among serum amylase, lipase, calcium levels. However, lymphocyte and platelets on comparing mean PLR. The hospital stay was also significantly more in ones who have PLR >300(23.30 ± 11.68) then 151-300 (11.95 ± 5.52) and ≤150(7.24 ± 4.43)

Table 19 (A): Properties of ROC curve in predicting organ failure

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
PLR0 hrs	0.794	0.050	0.001	0.697	0.891
PLR 24hrs	0.785	0.048	0.001	0.692	0.878
PLR 48hrs	0.838	0.043	0.001	0.753	0.923
mean PLR	0.810	0.045	0.001	0.721	0.899

The AUC was best for 48 hours (.838) for predicting complications of acute pancreatitis. It had STD error of .043 (.753 .923).

Table 19 (B): Predictive value at different cut offs at different time intervals

		Organ failure occurrence		Total	Chi-square value	p-value
		Yes	**No**			
PLR0HR 193.09	> 193.09	28	24	52	0.498	0.001
	< 193.09	6	77	83		
Total		**34**	**101**	**135**		
PLR 24HR 216.17	> 216.17	22	13	35	0.513	0.001
	< 216.17	12	88	100		
Total		**34**	**101**	**135**		
PLR 48Hr 206.01	> 206.01	24	10	34	0.607	0.001
	< 206.01	10	91	101		
Total		**34**	**101**	**135**		
mean PLR 206.37	< 206.37	24	15	39	0.531	0.001
	> 206.37	10	86	96		
Total		**34**	**101**	**135**		

Table 19 (C): Sensitivity, Specificity and Predictive value with respect to organ failure

	PLR 0HR 193.09	**PLR 24Hrs 216.17**	**PLR 48Hrs 206.01**	**Mean PLR 206.37**
Statistic	Value	Value	Value	Value
Sensitivity	82.35%	64.71%	70.59%	70.59%
Specificity	76.24%	87.13%	90.10%	85.15%
Positive Predictive Value (*)	53.85%	62.86%	70.59%	61.54%
Negative Predictive Value (*)	92.77%	88.00%	90.10%	89.58%
Accuracy (*)	77.78%	81.48%	85.19%	81.48%

We observed that predictive value and accuracy of PLR was best at 48 hours (85.19%) followed by mean and 24 hours (81.48%) and least at baseline (77.78%). However, the sensitivity was best at baseline (82.35%) followed by 24 hours that have cut off of 216.17 and sensitivity of 64.71% PLR of 206.01 at 48 hours was 70.59%. specific at 0,24,48 hours were

76.24%,87.13%,90.10% and 85.15% respectively. PPV was 53.85%,62.86%, 70.59%, and 61.54% at 0,24,48 and mean cut off respectively. PPV was 77.78%,81.48%, 85.19%, and 81.48% at 0,24,48 and mean cut off respectively.

At cut-off >216.16 It had sensitivity of 64.71%, specificity of 87.13%, PPV of 62.86%, NPV of 88% and overall accuracy of 81.48%.

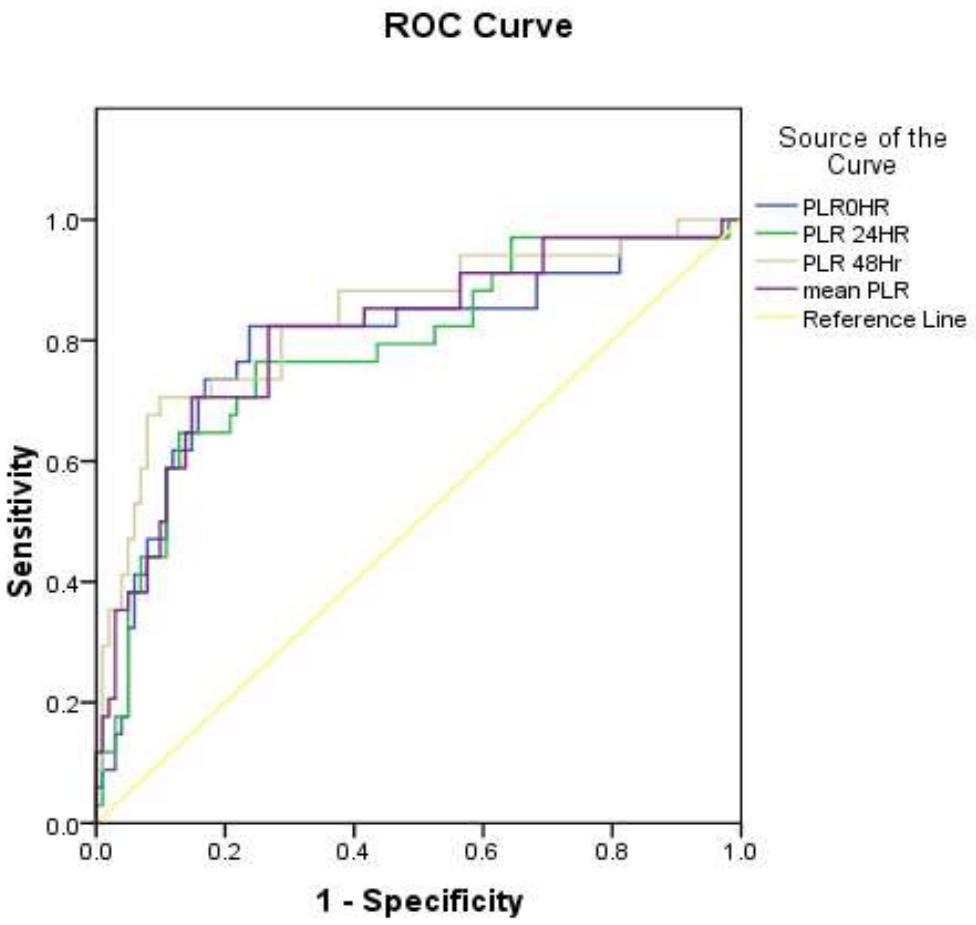

Figure 14: Receiver operative curve for PLR in predicting organ failure

Table 20(A) : Properties of ROC curve in predicting complications

Test Result Variable(s)	Area	Std. Error[a]	p-value	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
PLR0HR	.618	.050	.018	.521	.715
PLR 24hrs	.632	.048	.008	.537	.727
PLR 48hrs	.716	.044	.000	.629	.803
mean PLR	.652	.048	.002	.558	.746

The AUC was best for 48 hours (.716) for predicting complications of acute pancreatitis. it had STD error of .044 (.629-.803).

Table 20 (B): Predictive value at different cut offs at different time intervals

		Complications of acute pancreatitis		Total	Chi-square value	p-value
		Yes	**No**			
PLR0HR 185.62	> 185.62	38	20	58	0.327	0.001
	< 185.62	25	52	77		
Total		**63**	**72**	**135**		
PLR 24hrs 194.15	> 194.15	36	18	54	0.324	0.001
	< 194.15	27	54	81		
Total		**63**	**72**	**135**		
PLR 48hrs 170.14	> 170.14	40	19	59	0.373	0.001
	< 170.14	23	53	76		
Total		**63**	**72**	**135**		
mean PLR 178.90	> 178.90	40	19	59	0.373	0.001
	< 178.90	23	53	76		
Total		**63**	**72**	**135**		

Table 20 (C): Sensitivity, Specificity and Predictive value with respect to complications

	PLR0HR 185.62	**PLR 24HR 194.15**	**PLR 48Hr 170.14**	**mean PLR 178.90**
Statistic	**Value**	**Value**	**Value**	**Value**
Sensitivity	60.32%	57.14%	63.49%	63.49%
Specificity	72.22%	75.00%	73.61%	73.61%
Disease prevalence (*)	46.67%	46.67%	46.67%	46.67%
Positive Predictive Value (*)	65.52%	66.67%	67.80%	67.80%
Negative Predictive Value (*)	67.53%	66.67%	69.74%	69.74%
Accuracy (*)	66.67%	66.67%	68.89%	68.89%

PLR at 48 hours could predict complications (cut off 170.14) with accuracy of 68.89% had sensitivity for 63.49 specificity of 73.61, PPV if 67.80% and NPV of 69.74%.

At 24 hours it could predict complications (cut off 194.15) with accuracy of 66.67% had sensitivity of 57.14%, specificity of 75%, PPV and PPV of 66.67 %.

At 0 hours it could predict complications (cut off 185.62) with accuracy of 66.67% had sensitivity of 60.32%, specificity of 72.22%, PPV of 65.52% and PPV of 65.52%.

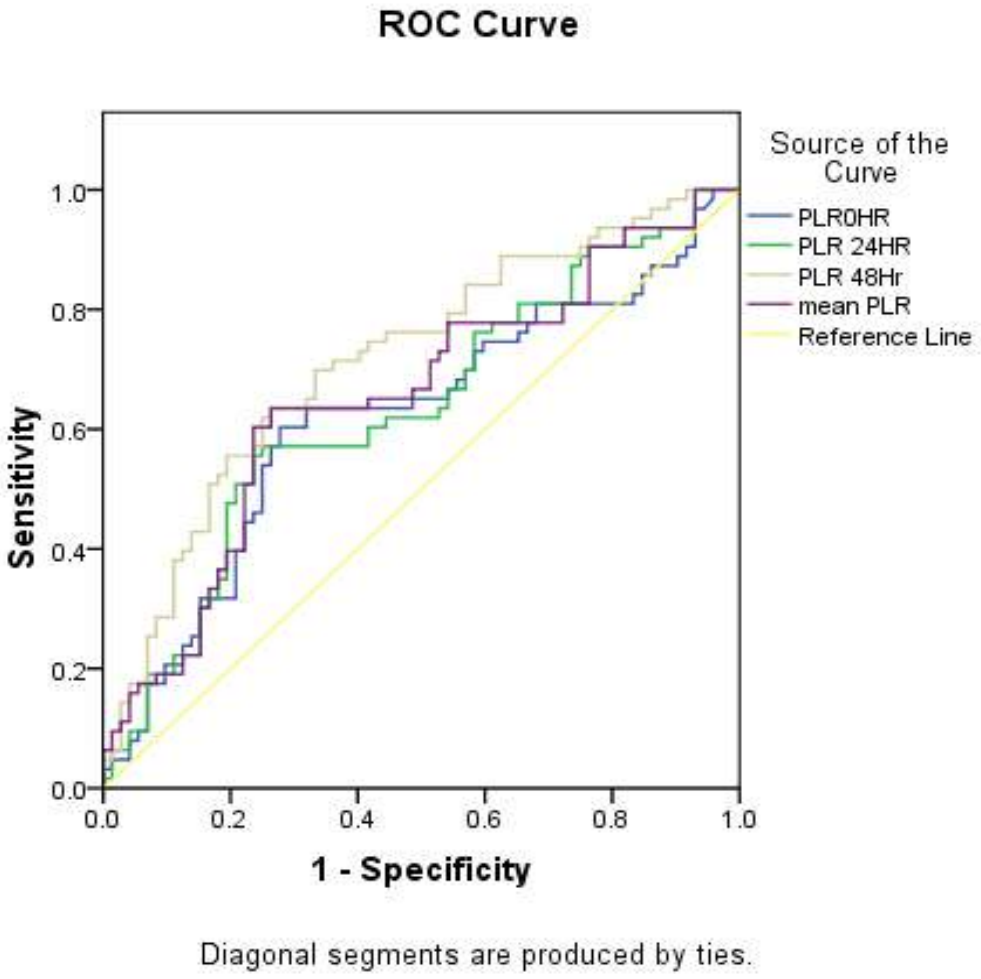

Figure 15: Receiver operative curve for PLR in predicting complications

Table 21(A): Properties of ROC for PLR in predicting ICU stay

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
PLR0HR	.650	.055	.005	.543	.758
PLR 24HR	.663	.053	.002	.560	.767
PLR 48Hr	.713	.049	.000	.617	.810
mean PLR	.679	.052	.001	.577	.781

The AUC was best for 48 hours (.713) for predicting need for ICU in case of acute pancreatitis. It had STD error of .049 (.617 -.810).

Table 21 (B): Predictive value at different cut offs at different time intervals

		Need of ICU and HDU stay		Total	Chi-square value	p-value
		Yes	No			
PLR0HR 203.64	> 203.64	26	18	44	0.380	0.001
	< 203.64	19	72	91		
Total		45	90	135		
PLR 24hrs 199.58	> 199.58	29	22	51	0.387	0.001
	< 199.58	16	68	84		
Total		45	90	135		
PLR 48hrs 206.01	> 206.01	23	11	34	0.414	0.001
	< 206.01	22	79	101		
Total		45	90	135		
mean PLR 178.90	> 178.90	32	27	59	0.381	0.001
	< 178.90	13	63	76		
Total		45	90	135		

Table 21 (C): Sensitivity, Specificity and Predictive value with respect to ICU stay

	PLR0HR 203.64	**PLR 24HR 199.58**	**PLR 48Hr 206.01**	**Mean PLR 178.90**
Sensitivity	57.78%	64.44%	51.11%	71.11%
Specificity	80.00%	75.56%	87.78%	70.00%
Positive Predictive Value (*)	59.09%	56.86%	67.65%	54.24%
Negative Predictive Value (*)	79.12%	80.95%	78.22%	82.89%
Accuracy (*)	72.59%	71.85%	75.56%	70.37%

PLR at 48 hours could predict complications (cut off 206.01) with accuracy of 75.56% had sensitivity for 51.11% specificity of 87.78%, PPV if 67.65% and NPV of 78.22%.

At 24 hours it could predict complications (cut off 199.58) with accuracy of 71.85% had sensitivity of 51.11%, specificity of 75.56 %, PPV of 56.86% and NPV of 80.95 % .

At 0 hours it could predict complications (cut off 203.64) with accuracy of 72.59% had sensitivity of 57.78%, specificity of 80%, PPV of 59.09% and NPV of 79.12%.

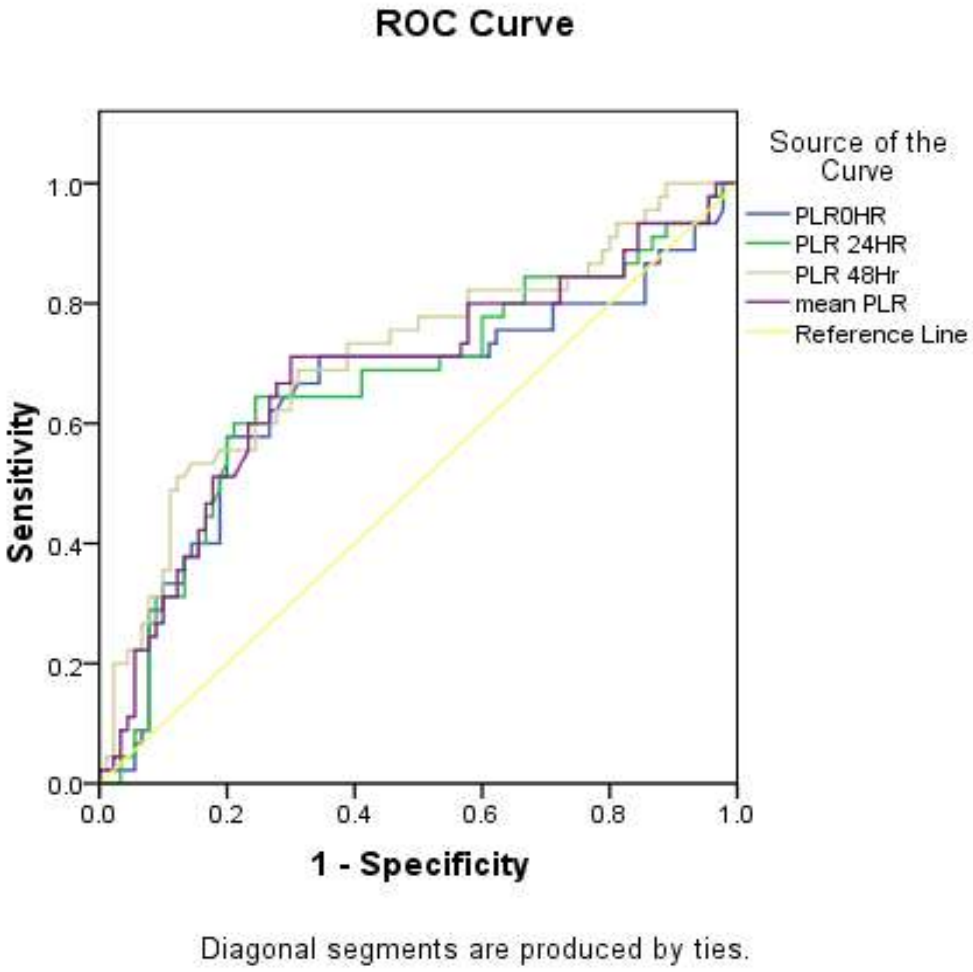

Figure 16: Receiver operative curve for PLR in predicting ICU stay

DISCUSSION

Acute Pancreatitis (AP) is defined as an acute condition presenting with abdominal pain, a threefold or greater rise in the serum levels of the pancreatic enzymes amylase or lipase, and/or characteristic findings of pancreatic inflammation on contrast enhanced CT.[1] It is a common inflammatory disease of the exocrine pancreas that causes multiple organ dysfunction and may lead to pancreatic necrosis and persistent organ failure, with a mortality of 1–5%.[56,57] Overall, it has a global incidence of 30–40 cases per 100,000 population per year and over twice that in some regions.[57,58] The incidence of acute pancreatitis in industrialized countries varies from 4.9 to 35 per 100,000 persons. In the United States, acute pancreatitis accounts for 327,474 hospitalizations and 2,834 deaths annually.[59,60] The incidence in children is not far behind at 10–15 cases per 100,000 children. The global incidence is rising, although studies suggest rates are currently more stable in Asia.[61]

It is a disease of variable severity in which some patients experience mild, self-limited attacks, while others manifest a severe, highly morbid and frequently lethal attack.[46] The clinical extent of AP varies widely from no symptoms to systemic inflammatory response syndrome (SIRS), persistent organ failure (POF), and death. The clinical presentation of AP is both unreliable and nonspecific and exhibits a sensitivity less than 40% for the prediction of adverse outcomes.[3]

Various severity scoring systems has been designed to assess the severity and predict prognosis. The Ranson score, the Acute Physiologic Assessment and Chronic Health Evaluation II (APACHE II) score, the Bedside Index for Severity in Acute Pancreatitis (BISAP) score, and the Glasgow-Imrie criteria are currently in wide use.[62] These scoring systems are time consuming and use many variables which are difficult to obtain in low resource settings.[1]

Inherited mutations in genes, encoding for digestive enzymes have been found in patients with a hereditary form of pancreatitis.[63,64] However, affected patients develop chronic, rather than acute pancreatitis. Therefore, in recent years, a novel concept has evolved, suggesting that systemic complications during AP result from uncontrolled activation of the inflammatory cascade. As indicated above, severe AP is associated with a significant mortality. Thus, early identification of severe forms of AP is crucial for outcome. In an attempt to identify surrogate parameters as predictors for severe AP, several association studies linking cytokines and chemokines with AP severity have been conducted.[65] Among these, serum levels of interleukin (IL)-6 and the IL-6-dependent acute phase protein, C-reactive protein (CRP) were identified as the most reliable predictors for severe AP.[63] But they are expensive and not readily available.[1]

Platelet to lymphocyte ratio (PLR) it can be derived from the complete blood count, is a novel index reflecting a systemic inflammatory burden that combines prognostic values of an individual's platelet and lymphocyte count. PLR has been shown to be an inflammatory marker and platelets are shown to be as active players in antimicrobial host defence and the induction of inflammation and tissue repair.[1] Increased NLR and PLR ratios have been associated with inflammatory conditions, and poor outcomes in severe AP. Various studies in the past have been done considering Neutrophil to Lymphocyte Ratio and its prognostic value in AP. Very few studies have been done on PLR. PLR is easily derivable value. Therefore, the present study was planned to evaluate the PLR value as a prognostic factor for the outcome of acute pancreatitis.

In our study, total 135 patients were taken. Patients were divided into 5 age groups. These were ≤ 30, 31-40, 41-50, 51-60 and > 60. In the present study, it was found that maximum patients were present in the age group 31-40 year (29.6%) followed by 41- 50 years (28.9%), 51-60 year

(8.1%) and >60 years with 22%, and least were in 51-60 years with 8%. the mean age of the patient was 43.50±13.72 years.

The results of the present study were in accordance with the study conducted by Kaushik MR et al (2017)[66] who studied 50 patients with acute pancreatitis and reported the mean (±SD) age of patients to be 43.74±16.85 years, with a range of 21 to 84 years. Katta VR in 2016[67] conducted a study on 102 patients in which the mean age was 46.31 ± 17.01 years. In both above studies the results were comparable to the present study.

In our study male predominance is seen. Out of total 135 patients, there were 93 males (68.9%) and 42 females (31.1%) in the study. Veena P et al[68] in 2020 90% were males and 10% were females. Males to females ratio was 9:1, in a study by Kaushik et al[66] 82% cases were male and 18% were women. 78.43% of our patients were males in a study by Katta et al[67] In a study by Arif et al[69] 39.3% patients were male while 60.7% patients were Female. The results of the present study were in accordance with the previous studies.

There are many etiological factors causing acute pancreatitis. These are gall stones, alcohol consumption, smoking, hypertriglyceridemia and drugs etc. In our study it was observed that, in maximum patients gall stone was the major etiological factor with 51.1% (69) cases, followed by 37% (50) cases that had alcohol as an etiologic agent. In 5.9% (8) cases hypercalcemia and idiopathic was the main reason for pancreatitis. The study done by cho SK (2018)[3] has found the similar results as found in our study with predominant etiological factor gall stone in majority of patients (n=134) followed by alcohol consumption (n= 109) and Elfatth et al. (2022)[54] also found gall stone (75.3%) as main etiological factor in their study. in a study by Januja et al[70] most common cause of AP was gallstones in 73 (68.9%), idiopathic in 20 (18.9%) patients and hypertriglyceridemia in 6 (5.7%) patients. However, idiopathic 5(10%) was

found to be the 3rd most common. In a study by Singh et al[71] there were high rate of post-endoscopic retrograde cholangiopancreatography (ERCP) and idiopathic acute pancreatitis (29.7%).

Organ failure

Organ failure was seen in 34(25.2%) patients while complications due to acute pancreatitis were seen in 63(46.7%). 45(33.3%) patients needed ICU. In a study by Chittipotula et al 2020[72] organ failure was seen in 52%, Venkatapuram MR et al.(2018)[73] showed it in 22%, Veena et al[68] in 22% while in a study by Katta et al[67] the organ failure was seen in almost all patients.

110(81.5%) patients were successfully treated and were discharged, 11% discharged against medical advice and 14(10.4%) were discharged on request

We compared the PLR ratio in patients with acute pancreatitis inn predicting the outcome of the patients. PLR ratio was calculated at different time intervals.

We observed that PLR showed that increase in PLR >300 was significantly associated with poor prognosis while <=150 was associated with better prognosis with respect to organ failure, complications of Acute pancreatitis, need for ICU and outcome of patient. The predictive value at 48 hours was best out of all the three-time intervals considered and was least at baseline however it was significant for predicting the organ failure and complications.

However, we observed that no significant difference was seen among serum amylase, lipase, calcium levels. However, lymphocyte and platelets at baseline, 24 hour, and 48 hours had significant difference among the three groups. The hospital stay was also significantly more in ones who

have PLR >300(17.71±8.84) then 151-300 (12.10± 7.02) and <=150(7.86±4.50).

The AUC was best for 48 hours (.838) for predicting organ failure of acute pancreatitis. it had STD error of .043 (.753-.923). At cut-off >216.16 It had sensitivity of 64.71%, specificity of 87.13%, PPV of 62.86%, NPV of 88% and overall accuracy of 81.48%.

The AUC was best for 48 hours (.716) for predicting complications of acute pancreatitis. it had STD error of .044 (.629-.803). PLR at 48 hours could predict complications (cut off 170.14) with accuracy of 68.89% had sensitivity for 63.49 specificity of 73.61, PPV if 67.80% and NPV of 69.74%.

The AUC was best for 48 hours (.713) for predicting need for ICU in case of acute pancreatitis. it had STD error of .049 (.617 -.810).

PLR at 48 hours could predict complications (cut off 206.01) with accuracy of 75.56% had sensitivity for 51.11% specificity of 87.78%, PPV if 67.65% and NPV of 78.22%.

Similar kind of finding were observed by Reddy et al1 they found that elevated PLR values were associated with increased frequency of ICU admissions, increased duration of hospital stay in survivors and a higher mortality. Therefore, higher PLR values are associated with poor prognosis in acute pancreatitis.

In this study, we were able to establish PLR as an prognostic factor in acute pancreatitis (AP). Platelets have an important role in coordinating inflammation and immune response. They affect inflammation by releasing cytokines, changing leukocyte and endothelial responses (18). PLR has been studied in various diseases (19-21). Platelet to lymphocyte ratio (PLR) is a relatively novel inflammatory marker that can be utilized for prognosis in various disease processes. Demir et al[44], investigated the

association between high PLR and mortality in patients with acute cardiogenic pulmonary edema and found out that high PLR group had significantly higher mortality rate than other groups. Various studies have shown PLR to predict the prognosis in patients with various inflammatory, ischemic and oncological disorders.

Ilhan et al[47] in their study on acute pancreatitis showed that to be a predictor in AP. However, comparison of PLR parameters of both the groups in their study was not statistically significant. Kaplan et al[51] in their study showed the threshold value of PLR level to be> 342.31 for predicting the severity of acute pancreatitis with 73.3% sensitivity and 99.2% specificity (AUC±SE=0.863± 0.077, p<0.001) Cho SK et al[3], investigated the relationship between NLR and PLR in the outcome in patients with Acute pancreatitis. They concluded that PLR can predict the severity of AP, but only in gallstone AP. The cut-off in their study was 229.1, with sensitivity and specificity of 70.4% and 52.3%.

In a study by Ekin et al[74], regardless of etiology, we found PLR to be significantly higher in the severe pancreatitis group. However, there was no alcohol in etiology of the patients included in the study.

To date, there are no previous studies which investigated the predictive value of PLR at the time of admission on outcomes in patients with AP. We investigated the role of PLR as a prognostic factor in predicting organ failure, complications and need of ICU at different time intervals for acute pancreatitis.

Kaplan et al[51] in their study found positive correlation between PLR and Ranson, Atlanta, BISAP scores and laboratory findings such as urea, creatinine, ESR, CRP and a cut-off value about PLR >342.31 was found to show disease severity with 73.3% sensitivity and 99.2% specificity.

Inspite of all the studies on prognostic markers of acute pancreatitis, it is still uncertain which scoring system or laboratory finding is the most

reliable in terms of determination of prognosis and mortality. In addition, each scoring system has its advantages disadvantages. Although the Ranson score presents a great advantage to assess disease severity, it is a disadvantage that it requires 48h data. Similarly, although it is very easy to use, the Atlanta classification is unable to differentiate between moderately acute pancreatitis and severe acute pancreatitis before 48 h after onset. Although BISAP provides quick data, it is complicated, cumbersome, and insufficiently sensitive. The PLR has advantages over other scoring systems such as being easy-to-use, simple, highly sensitive and specific and providing information regarding mortality and prognosis without requiring 48 hours of assessment time.

SUMMARY & CONCLUSION

This prospective study was conducted on 135 patients presenting with acute pancreatitis in Dayanand medical college and hospital to study the efficiency of platelet to lymphocyte ratio as marker to predict the outcome of acute pancreatitis. The results are summarised below:

- The mean age of the patient was 43.50±13.72 years. majority of the patients in present study were males (68.9%). In maximum patients gall stone was the major etiological factor with 51.1% (69) cases, followed by 37% (50) alcohol.
- Organ failure was seen in 34(25.2%) patients while complications due to acute pancreatitis were seen in 63(46.7%). 45(33.3%) patients needed ICU.
- We compared the PLR ratio in patients with acute pancreatitis inn predicting the outcome of the patients. PLR ratio was calculated at different time intervals.
- We observed that PLR showed that increase in PLR >300 was significantly associated with poor prognosis while <=150 was associated with better prognosis with respect to organ failure, complications of Acute pancreatitis, need for ICU and outcome of patient. The predictive value at 48 hours was best out of all the three-time intervals considered and was least at baseline however it was significant for predicting the organ failure and complications.
- The AUC was best for 48 hours (.838) for predicting organ failure of acute pancreatitis. it had STD error of .043 (.753-.923). At cut-off

>216.16 It had sensitivity of 64.71%, specificity of 87.13%, PPV of 62.86% ,NPV of 88% and overall accuracy of 81.48%.

- The AUC was best for 48 hours (.716) for predicting complications of acute pancreatitis. it had STD error of .044 (.629-.803). PLR at 48 hours could predict complications (cut off 170.14) with accuracy of 68.89% had sensitivity for 63.49 specificity of 73.61, PPV if 67.80% and NPV of 69.74%.
- The AUC was best for 48 hours (.713) for predicting need for ICU in case of acute pancreatitis. it had STD error of .049 (.617 -.810).
- PLR at 48 hours could predict complications (cut off 206.01) with accuracy of 75.56% had sensitivity for 51.11% specificity of 87.78%, PPV if 67.65% and NPV of 78.22%.

Conclusion

Inspite of all the studies on prognostic markers of acute pancreatitis, it is still uncertain which scoring system or laboratory finding is the most reliable in terms of determination of prognosis and mortality. In addition, each scoring system has its advantages disadvantages. Although the Ranson score presents a great advantage to assess disease severity, it is a disadvantage that it requires 48h data. Similarly, although it is very easy to use, the Atlanta classification is unable to differentiate between moderately acute pancreatitis and severe acute pancreatitis before 48 h after onset. Although BISAP provides quick data, it is complicated, cumbersome, and insufficiently sensitive. The PLR has advantages over other scoring systems such as being easy-to-use, simple, highly sensitive and specific and providing information regarding mortality and prognosis without requiring 48 hours of assessment time.

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PROFORMA

SR. NO. MRD NO.

NAME:	AGE/SEX:
ADDRESS:	OCCUPATION:
DATE OF ADMISSION:	DATE OF DISCHARGE:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST MEDICAL/ SURGICAL HISTORY:

PERSONAL HISTORY:

Marital status..... Dietary habits..... Smoking.....

Alcohol..... Substance abuse..... Other.....

FAMILY HISTORY:

BLOOD INVESTIGATIONS:

ON ADMISSION:	AT 24TH HOUR:	AT 48TH HOUR
PLATELET COUNT:	PLATELET COUNT:	PLATELET COUNT:
LYMPHOCYTE COUNT:	LYMPHOCYTE COUNT:	LYMPHOCYTE COUNT:
PLR RATIO:	PLR RATIO:	PLR RATIO:

DATE:					
HB					
TLC					
NEUTROPHIL COUNT					
MEAN PLATELET VOLUME					
HEMATOCRIT					
RDW					
BLOOD UREA					
SERUM CREATININE					
SERUM AMYLASE					
SERUM LIPASE					
SERUM CALCIUM					

LFT :

DATE:					
BILIRUBIN(T)					
BILIRUBIN(D)					
SGOT					
SGPT					
ALP					
TOTAL PROTEINS					
ALBUMIN					

ABG ANALYSIS:

DATE:					
pH					
Pco2					
Po2					
lactate					
Hct					
Hco3-					
Base excess					

GENERAL PHYSICAL EXAMINATION:

Appearance……. Sensorium…….

Vital signs: HR…… RR……. BP……. TEMP…….

Pallor……. Jaundice……. Cyanosis……. Edema…….

JVP……. Lymphadenopathy……. Clubbing…….

spo2(room air) ……. spo2(on oxygen) …….

GCS: E V M

Pupillary size (L) ……. (R) ……. Reaction(L) ……. (R) …….

ANTHROPOMETEY:

weight(kg) ……. height(m) ……. BMI…….

Build……. Anemia……. vitamin deficiency…….

nutrition status…….

RADIOLOGICAL INVESTIGATIONS (USG/CT/MRI):

ABG ANALYSIS:

DATE:					
pH					
Pco2					
Po2					
lactate					
Hct					
Hco3-					
Base excess					

GENERAL PHYSICAL EXAMINATION:

Appearance……. Sensorium…….

Vital signs: HR…… RR……. BP……. TEMP…….

Pallor……. Jaundice……. Cyanosis……. Edema…….

JVP……. Lymphadenopathy……. Clubbing…….

spo2(room air) ……. spo2(on oxygen) …….

GCS: E V M

Pupillary size (L) ……. (R) ……. Reaction(L) ……. (R) …….

ANTHROPOMETEY:

weight(kg) ……. height(m) ……. BMI…….

Build……. Anemia……. vitamin deficiency…….

nutrition status…….

RADIOLOGICAL INVESTIGATIONS (USG/CT/MRI):

SYSTEMIC EXAMINATION:

ABDOMEN:

RESPIRATORY:

CVS:

CNS:

ETIOLOGY OF ACUTE PANCREATITIS:

FINAL OUTCOME:

LENGTH OF HOSPITAL STAY	
NEED OF ICU/HDU STAY	
DOR/DEATH/DAMA/discharge	
TYPE OF MANAGEMENT (MEDICAL/PCD/SURGICAL)	
ORGAN FAILURE OCCURRENCE/ MODS	
COMPLICATIONS OF ACUTE PANCREATITIS	

SIGNATURE OF CANDIDATE: **SIGNATURE OF SUPERVISOR:**

KEY TO MASTER CHART

Cause of pancreatitis
• 1 – Biliary
• 2 – Alcohol
• 3 – Hypercalcemia
• 4 – Idiopathic
Age group
• ≤30
• 31 – 40
• 41 – 50
• 51 – 60
• >60
Distribution of patients according to PLR ratio
• 1 – ≤150
• 2 – 151-300
• 3 – >300
Type of management
• 1 – Conservative
• 2 – Percutaneous catheter drainage
• 3 – Surgical
Complications of pancreatitis
• 0 – No
• 1 – Yes
Need of ICU and HDU stay
• 0 – No
• 1 – Yes
DOR, Death, DAMA, Discharge
• 1 – Discharge
• 2 – DAMA
• 3 – DOR
• 4 – Death

S0	NAME	AGE	Age group	SEX	MRD0	cause of pancreatitis	SERUM AMYLASE	SERUM LIPASE	SERUM CALCIUM	LYMPHOCYTE 0hr	LYMPHOCYTEAT 24HOUR
1.00	A S	33	2	Male	1201956	1	950	1210	9.10	1200	1190
2.00	A k	37	2	Female	1244947	2	295	300	4.10	673	668
3.00	B D	72	5	Female	1202504	2	550	920	8.90	890	880
4.00	F D	45	3	Male	1251404	2	350	250	8.80	450	440
5.00	G S	55	4	Male	1178281	3	333	830	8.40	2340	2330
6.00	H S	35	2	Male	701798	4	445	842	8.60	1200	1150
7.00	J S	29	1	Male	1200310	3	440	775	9.10	880	870
8.00	J S	75	5	Male	1192371	1	330	650	8.90	780	750
9.00	J S	48	3	Male	1217035	1	450	852	9.10	700	869
10.00	J S	42	3	Male	1117752	1	353	654	8.80	899	775
11.00	M	32	2	Male	1159777	3	301	856	9.10	620	610
12.00	R K	42	3	Male	1205466	1	299	550	8.20	1230	1220
13.00	P K	41	3	Male	899988	4	356	954	9.20	1800	1790
14.00	P R	88	5	Female	1219667	2	546	1013	9.50	700	690
15.00	R S	55	4	Male	1158765	1	344	956	8.20	1026	1000
16.00	R	38	2	Male	1202513	2	414	985	8.40	1836	1886
17.00	R S	28	1	Male	830597	1	256	578	8.50	1700	1690
18.00	R S	31	2	Female	1202570	2	333	564	8.40	2000	2150
19.00	S S	49	3	Male	1134544	1	412	865	7.10	1200	1100
20.00	S S	56	4	Male	1129016	1	235	565	8.80	1500	1490
21.00	R K	52	4	Male	1196453	2	254	457	8.20	1450	1440
22.00	M	30	1	Female	1201943	2	289	498	8.90	1176	1326
23.00	H D S	36	2	Male	1192072	1	249	564	8.60	1420	1570
24.00	R k	45	3	Male	1010569	1	259	543	8.80	1230	1220
25.00	D S	34	2	Male	1172510	2	286	427	8.70	1432	1422
26.00	B K	79	5	Female	1222060	2	258	428	8.20	1980	1970
27.00	S	50	3	Female	5053	2	236	568	8.30	1534	1524
28.00	P S	58	4	Female	1114852	2	295	300	4.10	801	951
29.00	M S	41	3	Male	1124397	1	350	250	8.80	1700	1690
30.00	S S	34	2	Male	1145778	2	333	830	8.40	1560	1550
31.00	N	58	4	Female	1201742	2	445	842	8.60	1400	1390
32.00	S K	24	1	Male	1184037	4	299	550	8.20	3276	3426
33.00	S K	55	4	Male	1220471	1	356	954	9.20	1800	1790
34.00	T P	64	5	Male	1099386	2	254	985	8.20	900	890
35.00	T G	39	2	Male	1131468	2	414	985	8.40	1900	1890
37.00	J	74	5	Male	561475	2	256	578	8.50	1700	1690
38.00	R J	18	1	Male	1145452	2	333	564	8.40	1392	1542
39.00	B S	36	2	Male	1056553	1	235	565	8.80	1040	1190
40.00	C S	35	2	Male	1258458	1	254	457	8.20	2400	2390
41.00	R K	32	2	Female	1046595	2	289	498	8.90	890	880
42.00	G k	55	4	Female	1049834	2	249	564	8.60	1960	1950
43.00	M S	50	3	Male	1050471	1	356	612	8.90	1090	1000
44.00	C D	55	4	Female	1065696	2	259	543	8.80	1980	1970
45.00	M K	33	2	Female	1130571	2	286	427	8.70	2736	2586
46.00	A S	49	3	Male	1201956	1	258	428	8.20	1440	1430
47.00	S L	43	3	Male	1053584	2	236	568	8.30	1024	1014
48.00	S S	21	1	Female	1179742	2	356	954	9.20	1800	1950
49.00	J S	44	3	Male	1169260	2	359	869	9.10	839	829
49.00	B B	44	3	Male	1160461	2	359	869	9.10	839	829
50.00	k S	60	4	Male	958967	1	414	985	8.40	1450	1600
51.00	A S	37	2	Male	1164363	1	256	578	8.50	1760	1750
52.00	D S	34	2	Male	1149757	2	333	564	8.40	1040	1030
53.00	M S	33	2	Male	34601	1	425	1023	9.10	1592	1400
54.00	M S	36	2	Male	114859	1	480	1189	8.80	1525	1256
55.00	R S	46	3	Male	1169853	2	235	565	8.80	1450	1440
56.00	M M	44	3	Female	1181840	2	254	457	8.20	1600	1590
57.00	G S	44	3	Male	1179281	1	398	654	8.90	1200	1150
58.00	G S	41	3	Male	1279281	1	378	689	8.80	1300	1250
59.00	S	52	4	Female	1183179	2	289	498	8.90	1230	1220
60.00	V S	45	3	Female	1186776	2	356	548	8.80	1500	1490
61.00	V S	48	3	Female	1045848	2	348	588	8.70	1400	1455
62.00	G S	25	1	Male	1245852	1	369	654	8.90	1612	1400
63.00	G S	22	1	Male	1045757	1	359	689	8.80	1632	1390
64.00	S S	55	4	Male	1045128	1	357	698	8.70	900	935
65.00	S S	59	4	Male	1145842	1	389	710	8.60	890	980
66.00	S S	32	2	Male	1238772	1	256	564	8.90	1395	1265
67.00	S S	35	2	Male	1038772	1	278	542	8.70	1358	1245
68.00	B M K	55	4	Male	1216548	2	458	756	9.10	1160	1150

S0	NAME	LYMPHOCYTEAT 48HOUR	PLTS 0HR	PLTS 24Hr	PLTS48hr	PLR0HR	PLR 24HR	PLR 48Hr	mean PLR	@0cat	@24catPLR
1.00	A S	1200	252000	248000	247800	210.00	208.40	206.50	208.30	2	2
2.00	A k	680	142000	143000	149000	211.00	214.07	219.12	214.73	2	2
3.00	B D	900	352000	350000	352000	395.51	397.73	391.11	394.78	3	3
4.00	F D	1147	180000	165000	177000	400.00	355.60	154.32	303.31	3	3
5.00	G S	2580	340000	345000	367000	145.30	140.24	142.25	142.60	1	1
6.00	H S	1436	251000	241000	251000	209.17	209.57	174.79	197.84	2	2
7.00	J S	850	181000	180000	184000	205.68	206.90	216.47	209.68	2	2
8.00	J S	720	180000	179000	181000	230.77	238.67	251.39	240.27	2	2
9.00	J S	1100	296000	295000	297000	422.86	339.47	270.00	344.11	3	3
10.00	J S	700	250000	249000	251000	278.09	321.29	358.57	319.32	2	3
11.00	M	960	250000	251200	252400	403.23	411.80	262.92	359.32	3	3
12.00	R K	1300	234000	256000	263000	190.24	209.84	202.31	200.80	2	2
13.00	P K	1800	278500	279700	282900	154.72	156.26	157.17	156.05	2	2
14.00	P R	900	195000	198000	262000	278.57	286.96	291.11	285.55	2	2
15.00	R S	920	93000	92000	94000	90.64	92.00	102.17	94.94	1	1
16.00	R	1906	260000	250000	251200	141.61	132.56	131.79	135.32	1	1
17.00	R S	1700	345000	346200	349400	202.94	204.85	205.53	204.44	2	2
18.00	R S	2470	230000	250000	251200	115.00	116.28	101.70	110.99	1	1
19.00	S S	1400	327000	326000	328000	272.50	296.36	234.29	267.72	2	2
20.00	S S	1500	378000	379200	382400	252.00	254.50	254.93	253.81	2	2
21.00	R K	1600	214000	226000	238000	147.59	156.94	148.75	151.09	1	2
22.00	M	1646	203000	250000	251200	172.62	188.54	152.61	171.26	2	2
23.00	H D S	1890	311000	250000	251200	219.01	159.24	132.91	170.39	2	2
24.00	R k	1300	230000	231200	234400	186.99	189.51	180.31	185.60	2	2
25.00	D S	1450	212000	213200	216400	148.04	149.93	149.24	149.07	1	1
26.00	B K	1950	280000	281200	284400	141.41	142.74	145.85	143.33	1	1
27.00	S	1500	249000	250200	253400	162.32	164.17	168.93	165.14	2	2
28.00	P S	1271	130000	180000	181200	162.30	189.27	142.56	164.71	2	2
29.00	M S	1700	213000	214200	217400	125.29	126.75	127.88	126.64	1	1
30.00	S S	1710	234000	246000	258000	150.00	158.71	150.88	153.20	1	2
31.00	N	1400	296000	300000	290000	211.43	215.83	207.14	211.47	2	2
32.00	S K	3746	224000	250000	251200	68.38	72.97	67.06	69.47	1	1
33.00	S K	1950	234500	246500	258500	130.28	137.71	132.56	133.52	1	1
34.00	T P	900	197100	200100	204100	219.00	224.83	226.78	223.54	2	2
35.00	T G	2050	324000	336000	348000	170.53	177.78	169.76	172.69	2	2
37.00	J	1850	300000	312000	324000	176.47	171.43	175.14	174.34	2	2
38.00	R J	1862	188000	190000	191200	135.06	123.22	102.69	120.32	1	1
39.00	B S	1510	144000	150000	151200	138.46	126.05	100.13	121.55	1	1
40.00	C S	2550	290000	302000	314000	120.83	126.36	123.14	123.44	1	1
41.00	R K	900	234000	235200	238400	262.92	267.27	264.89	265.03	2	2
42.00	G k	1710	255000	256200	259400	130.10	131.38	151.70	137.73	1	1
43.00	M S	1100	380000	379000	381000	348.62	379.00	346.36	358.00	3	3
44.00	C D	2830	365000	366200	369400	184.34	185.89	130.53	166.92	2	2
45.00	M K	2906	421000	410000	411200	153.87	158.55	141.50	151.31	2	2
46.00	A S	2290	254000	255200	258400	176.39	178.46	112.84	155.90	2	2
47.00	S L	1874	325000	327000	330200	317.38	322.49	176.20	272.02	3	3
48.00	S S	2000	180000	210000	211200	100.00	107.69	105.60	104.43	1	1
49.00	J S	900	260000	250000	255000	309.89	301.57	283.33	298.26	3	3
49.00	B B	900	260000	250000	255000	309.89	301.57	283.33	298.26	3	3
50.00	k S	1920	200000	250000	251200	137.93	156.25	130.83	141.67	1	2
51.00	A S	2610	316000	318000	321200	179.55	181.71	123.07	161.44	2	2
52.00	D S	1100	144000	146000	149200	138.46	141.75	135.64	138.62	1	1
53.00	M S	1500	206000	205000	207000	129.40	146.43	138.00	137.94	1	1
54.00	M S	1460	205000	206000	201500	128.40	141.43	138.00	135.94	1	1
55.00	R S	2300	241000	243000	246200	166.21	168.75	107.04	147.33	2	2
56.00	M M	1600	318000	308900	305000	198.75	194.28	190.63	194.55	2	2
57.00	G S	1200	250000	249000	251000	208.33	216.52	209.17	211.34	2	2
58.00	G S	1260	260000	248000	254000	210.33	236.52	220.17	231.34	2	2
59.00	S	1300	234000	256000	263000	190.24	209.84	202.31	200.80	2	2
60.00	V S	1680	290000	300000	290000	193.33	201.34	172.62	189.10	2	2
61.00	V S	1670	280000	290000	285000	192.33	208.34	171.62	187.10	2	2
62.00	G S	1300	225000	219000	200000	139.58	156.43	153.85	149.95	1	2
63.00	G S	1310	235000	228000	200000	135.58	146.43	155.85	146.95	1	2
64.00	S S	930	231000	230000	232000	256.67	245.99	249.46	250.71	2	2
65.00	S S	890	241000	220000	242000	246.67	256.99	265.46	240.71	2	2
66.00	S S	1300	344000	343000	345000	246.59	271.15	265.38	261.04	2	2
67.00	S S	1345	345000	342000	344000	236.59	261.15	266.38	271.04	2	2
68.00	B M K	1700	340000	360000	375000	293.10	313.04	220.59	275.58	2	3

S0	NAME	@48catPLR	mean	LENGTH OF HOSPITAL STAY	TYPE OF MANAGEMENT	ORGAN FAILURE OCCURRENCE	COMPLICATIONS OF ACUTE PANCREATITIS	NEED OF ICU AND HDU STAY	DOR DEATH DAMA DISCHARGE	
1.00	A S	2	2	14	2	1	1	1	DOR	3
2.00	A k	2	2	25	2	0	1	1	discharge	1
3.00	B D	3	3	16	2	1	1	0	discharge	1
4.00	F D	2	3	18	1	0	0	0	discharge	1
5.00	G S	1	1	3	1	0	0	0	DAMA	2
6.00	H S	2	2	9	2	0	1	1	discharge	1
7.00	J S	2	2	20	2	1	1	1	discharge	1
8.00	J S	2	2	15	1	1	1	1	discharge	1
9.00	J S	2	3	41	1	1	1	1	discharge	1
10.00	J S	3	3	47	2	1	1	1	DAMA	2
11.00	M	2	3	23	1	1	1	0	discharge	1
12.00	R K	2	2	10	2	0	1	0	discharge	1
13.00	P K	2	2	9	1	0	1	0	discharge	1
14.00	P R	2	2	27	1	1	0	1	discharge	1
15.00	R S	1	1	23	1	1	0	1	discharge	1
16.00	R	1	1	3	1	0	1	0	DAMA	2
17.00	R S	2	2	10	1	0	0	0	discharge	1
18.00	R S	1	1	4	2	0	1	0	DOR	3
19.00	S S	2	2	11	1	1	1	1	DAMA	2
20.00	S S	2	2	6	1	0	0	0	DOR	3
21.00	R K	1	2	14	1	0	1	1	discharge	1
22.00	M	2	2	9	1	0	0	0	discharge	1
23.00	H D S	1	2	4	1	0	0	0	discharge	1
24.00	R k	2	2	10	1	0	0	0	discharge	1
25.00	D S	1	1	9	1	0	0	0	discharge	1
26.00	B K	1	1	9	1	0	0	0	discharge	1
27.00	S	2	2	9	1	0	0	0	discharge	1
28.00	P S	1	2	6	1	0	0	0	DOR	3
29.00	M S	1	1	3	1	0	0	0	discharge	1
30.00	S S	2	2	14	1	0	1	0	DOR	3
31.00	N	2	2	9	1	0	0	0	discharge	1
32.00	S K	1	1	4	1	0	0	0	discharge	1
33.00	S K	1	1	15	1	0	1	0	discharge	1
34.00	T P	2	2	8	1	1	0	0	discharge	1
35.00	T G	2	2	8	1	0	0	0	discharge	1
37.00	J	2	2	8	1	0	0	0	discharge	1
38.00	R J	1	1	5	1	0	0	0	discharge	1
39.00	B S	1	1	3	1	0	0	0	discharge	1
40.00	C S	1	1	11	1	0	1	1	discharge	1
41.00	R K	2	2	5	1	0	0	0	discharge	1
42.00	G k	2	1	5	1	0	0	0	discharge	1
43.00	M S	3	3	14	1	1	1	1	discharge	1
44.00	C D	1	2	7	1	0	0	0	discharge	1
45.00	M K	1	2	9	1	0	0	0	discharge	1
46.00	A S	1	2	8	1	0	0	0	discharge	1
47.00	S L	2	2	7	1	0	0	0	discharge	1
48.00	S S	1	1	9	1	0	1	1	discharge	1
49.00	J S	2	2	20	3	1	1	1	discharge	1
49.00	B B	2	2	20	3	1	1	1	discharge	1
50.00	k S	1	1	5	1	0	0	0	discharge	1
51.00	A S	1	2	9	1	0	0	0	discharge	1
52.00	D S	1	1	7	1	0	0	0	discharge	1
53.00	M S	1	1	15	2	1	1	1	discharge	1
54.00	M S	1	1	15	2	1	1	1	discharge	1
55.00	R S	1	1	5	1	0	0	0	discharge	1
56.00	M M	2	2	10	1	0	1	0	discharge	1
57.00	G S	2	2	17	1	1	1	1	discharge	1
58.00	G S	2	2	17	1	1	1	1	discharge	1
59.00	S	2	2	17	1	0	1	1	discharge	1
60.00	V S	2	2	16	1	1	1	1	discharge	1
61.00	V S	2	2	16	1	1	1	1	discharge	1
62.00	G S	2	1	10	1	1	1	1	discharge	1
63.00	G S	2	1	10	1	1	1	1	discharge	1
64.00	S S	2	2	15	1	1	1	1	discharge	1
65.00	S S	2	2	15	1	1	1	1	discharge	1
66.00	S S	2	2	14	1	1	1	1	discharge	1
67.00	S S	2	2	14	1	1	1	1	discharge	1
68.00	B M K	2	2	27	3	1	1	1	DOR	3

S0	NAME	AGE	Age group	SEX	MRD0	cause of pancreatitis	SERUM AMYLASE	SERUM LIPASE	SERUM CALCIUM	LYMPHOCYTE 0hr	LYMPHOCYTEAT 24HOUR
69.00	K K	35	2	Female	1230383	2	249	564	8.60	1760	1750
70.00	M S	55	4	Male	1133649	2	259	543	8.80	1500	1650
71.00	S k	36	2	Male	1240015	2	356	598	8.90	1255	1245
72.00	R k	45	3	Male	985478	1	286	427	8.70	1700	1700
73.00	B k	48	3	Male	1198624	1	258	428	8.20	1800	1790
74.00	G S	46	3	Male	1171824	3	236	568	8.30	1400	1390
75.00	H k	44	3	Female	1249494	2	295	300	4.10	1240	1230
76.00	k R	54	4	Female	1110436	2	350	250	8.80	2100	2250
77.00	k k	63	5	Male	1061868	1	333	830	8.40	2190	2340
78.00	K S	41	3	Male	75986	1	445	842	8.60	1560	1550
79.00	L U	30	1	Male	1121870	2	299	550	8.20	1850	1840
80.00	M k	75	5	Female	1222699	2	356	653	9.10	1200	1190
81.00	N	30	1	Female	1205454	4	356	785	9.20	730	720
82.00	M K	46	3	Female	1130571	3	356	954	9.20	1500	1490
83.00	M D	77	5	Female	1232589	4	235	565	8.80	2090	2080
84.00	N	23	1	Female	1026569	3	356	689	8.90	760	750
85.00	S K	25	1	Male	1133018	2	254	457	8.20	1400	1410
86.00	R K	25	1	Male	1238136	1	354	756	9.20	1200	2300
87.00	R S	40	2	Male	1138788	1	324	547	8.80	900	750
88.00	S k	53	4	Male	1082461	1	289	498	8.90	1850	1840
89.00	N A	35	2	Female	1013324	2	389	599	8.90	1230	1220
90.00	N	37	2	Female	1205454	2	353	654	8.8	1270	1210
91.00	L	45	3	Male	1121870	2	301	856	9.1	693	638
92.00	I D T	55	4	Male	1118681	3	345	845	9.2	470	410
93.00	P	35	2	Male	1141844	4	299	550	8.2	2360	2300
94.00	A S	42	3	Male	1118347	1	356	954	9.2	1220	1120
95.00	J p	41	3	Male	1118836	4	546	1013	9.5	1250	1190
96.00	S D	38	2	Male	1113228	2	344	956	8.2	1820	1760
97.00	JS	28	1	Male	1217828	1	414	985	8.4	1856	1856
98.00	M	31	2	Female	1119017	2	256	578	8.5	1720	1660
99.00	DR	56	4	Male	1036655	1	333	564	8.4	2020	2120
100.00	AS	52	4	Male	1128326	2	412	865	7.1	1520	1460
101.00	B S	30	1	Female	1109240	2	235	565	8.8	1470	1410
102.00	H P S	36	2	Male	1188692	1	254	457	8.2	1196	1296
103.00	R S	45	3	Male	1181001	1	289	498	8.9	1440	1540
104.00	S S	34	2	Male	1145778	2	249	564	8.6	1250	1190
105.00	B k	79	5	Female	1222060	2	259	543	8.8	1452	1392
106.00	S	50	3	Female	1229552	2	286	427	8.7	2000	1940
107.00	k k	58	4	Female	1137673	2	258	428	8.2	1554	1494
108.00	TPS	41	3	Male	1315567	1	236	568	8.3	821	921
109.00	V G	34	2	Male	1118351	2	295	300	4.1	1720	1660
110.00	SK	58	4	Female	1206046	2	350	250	8.8	1580	1520
111.00	S G	24	1	Male	1050987	4	333	830	8.4	1420	1360
112.00	B S	55	4	Male	833125	1	445	842	8.6	3296	3396
113.00	R K	39	2	Male	1059376	2	299	550	8.2	1820	1760
114.00	M L	74	5	Male	1311118	2	356	954	9.2	1920	1860
115.00	A G	18	1	Male	853981	2	254	985	8.2	1720	1660
116.00	S S	36	2	Male	1222836	1	414	985	8.4	1412	1512
117.00	V S	35	2	Male	595127	1	256	578	8.5	1060	1160
118.00	U R	32	2	Female	1217059	2	333	564	8.4	2420	2360
119.00	R R	55	4	Female	1135214	2	235	565	8.8	910	850
120.00	P S	55	4	Female	1114852	2	254	457	8.2	1980	1920
121.00	P	33	2	Female	1083310	2	289	498	8.9	2000	1940
122.00	N S	49	3	Male	102322	1	249	564	8.6	2756	2556
123.00	S S	43	3	Male	74031	2	356	612	8.9	1460	1400
124.00	A	21	1	Female	1059471	2	259	543	8.8	1044	984
125.00	R S	60	4	Male	969216	1	286	427	8.7	1820	1920
126.00	V S	37	2	Male	1198606	1	258	428	8.2	1470	1570
127.00	S A	34	2	Male	874930	2	236	568	8.3	1780	1720
128.00	P k	46	3	Male	1097671	2	356	954	9.2	1060	1000
129.00	J k	44	3	Female	1230257	2	359	869	9.1	1470	1410
130.00	S D	52	4	Female	1060348	2	359	869	9.1	1620	1560
131.00	S G	35	2	Female	1036192	2	414	985	8.4	1250	1190
132.00	G S	31	2	Male	1311099	2	256	578	8.5	1780	1720
133.00	T A	45	3	Male	1021573	1	333	564	8.4	1520	1620
134.00	RRG	48	3	Male	1024779	1	425	1023	9.1	1720	1670
135.00	HS	46	3	Male	1242731	3	425	1023	9.1	1820	1760

S0	NAME	LYMPHOCYTEAT 48HOUR	PLTS 0HR	PLTS 24Hr	PLTS48hr	PLR0HR	PLR 24HR	PLR 48Hr	mean PLR	@0cat	@24catPLR
69.00	K K	2010	254000	256000	259200	144.32	146.29	128.96	139.85	1	1
70.00	M S	1970	200000	210000	211200	133.33	127.27	107.21	122.60	1	1
71.00	S k	1350	250000	215000	260000	199.20	172.69	192.59	188.16	2	2
72.00	R k	1720	219000	220000	221200	128.82	129.41	128.60	128.95	1	1
73.00	B k	2000	225000	245000	265000	125.00	136.87	132.50	131.46	1	1
74.00	G S	1550	270000	282000	294000	192.86	202.88	189.68	195.14	2	2
75.00	H k	1100	450000	420000	390000	362.90	341.46	354.55	352.97	3	3
76.00	k R	2570	210000	210000	211200	100.00	93.33	82.18	91.84	1	1
77.00	k k	2660	240000	230000	231200	109.59	98.29	86.92	98.27	1	1
78.00	K S	2210	210000	212000	215200	134.62	136.77	97.38	122.92	1	1
79.00	L U	1780	334000	330000	320000	180.54	179.35	179.78	179.89	2	2
80.00	M k	1100	195000	190000	190000	162.50	159.66	172.73	164.96	2	2
81.00	N	970	230000	245000	250000	315.07	340.28	257.73	304.36	3	3
82.00	M K	1740	425000	413000	400000	283.33	277.18	229.89	263.47	2	2
83.00	M D	2100	254000	256000	259200	121.53	123.08	123.43	122.68	1	1
84.00	N	850	234000	233500	214500	307.89	311.33	252.35	290.53	3	3
85.00	S K	1400	351000	350000	351200	250.71	248.23	250.86	249.93	2	2
86.00	R K	3000	350000	349000	351000	291.67	151.74	117.00	186.80	2	2
87.00	R S	523	290000	289000	291000	322.22	385.33	556.41	421.32	3	3
88.00	S k	1780	334000	330000	320000	180.54	179.35	179.78	179.89	2	2
89.00	N A	1100	295000	290000	262000	239.84	237.70	238.18	238.57	2	2
90.00	N	1280	255600	249700	254000	201.2598425	206.3636364	198.4375	202.0203263	2	2
91.00	L	710	141600	142700	148000	204.3290043	223.6677116	208.4507042	212.1491401	2	2
92.00	I D T	1177	179600	164700	176000	382.1276596	401.7073171	149.5327103	311.1225623	3	3
93.00	P	2610	339600	344700	366000	143.8983051	149.8695652	140.2298851	144.6659185	1	1
94.00	A S	1466	250600	240700	250000	205.4098361	214.9107143	170.53206	196.9508701	2	2
95.00	J p	1330	233600	255700	262000	186.88	214.8739496	196.9924812	199.5821436	2	2
96.00	S D	1830	278100	279400	281900	152.8021978	158.75	154.0437158	155.1986379	2	2
97.00	JS	1936	259600	249700	250200	139.8706897	134.5366379	129.2355372	134.5476216	1	1
98.00	M	1730	344600	345900	348400	200.3488372	208.373494	201.3872832	203.3698715	2	2
99.00	DR	2500	229600	249700	250200	113.6633663	117.7830189	100.08	110.5087951	1	1
100.00	AS	1530	377600	378900	381400	248.4210526	259.5205479	249.2810458	252.4075488	2	2
101.00	B S	1630	213600	225700	237000	145.3061224	160.070922	145.398773	150.2586058	1	2
102.00	H P S	1676	202600	249700	250200	169.3979933	192.6697531	149.2840095	170.4505853	2	2
103.00	R S	1920	310600	249700	250200	215.6944444	162.1428571	130.3125	169.3832672	2	2
104.00	S S	1330	229600	230900	233400	183.68	194.0336134	175.4887218	184.4007784	2	2
105.00	B k	1480	211600	212900	215400	145.7300275	152.9454023	145.5405405	148.0719901	1	2
106.00	S	1980	279600	280900	283400	139.8	144.7938144	143.1313131	142.5750425	1	1
107.00	k k	1530	248600	249900	252400	159.97426	167.2690763	164.9673203	164.0702188	2	2
108.00	TPS	1301	129600	179700	180200	157.8562728	195.1140065	138.5088394	163.8263729	2	2
109.00	V G	1730	212600	213900	216400	123.6046512	128.8554217	125.0867052	125.848926	1	1
110.00	SK	1740	233600	245700	257000	147.8481013	161.6447368	147.7011494	152.3979958	1	2
111.00	S G	1430	295600	299700	289000	208.1690141	220.3676471	202.0979021	210.2115211	2	2
112.00	B S	3776	223600	249700	250200	67.83980583	73.52767962	66.26059322	69.20935956	1	1
113.00	R K	1980	234100	246200	257500	128.6263736	139.8863636	130.0505051	132.8544141	1	1
114.00	M L	2080	323600	335700	347000	168.5416667	180.483871	166.8269231	171.9508202	2	2
115.00	A G	1880	299600	311700	323000	174.1860465	187.7710843	171.8085106	177.9218805	2	2
116.00	S S	1892	187600	189700	190200	132.8611898	125.462963	100.5285412	119.6175647	1	1
117.00	V S	1540	143600	149700	150200	135.4716981	129.0517241	97.53246753	120.6852966	1	1
118.00	U R	2580	289600	301700	313000	119.6694215	127.8389831	121.3178295	122.942078	1	1
119.00	R R	930	233600	234900	237400	256.7032967	276.3529412	255.2688172	262.7750184	2	2
120.00	P S	1740	254600	255900	258400	128.5858586	133.28125	148.5057471	136.7909519	1	1
121.00	P	2860	364600	365900	368400	182.3	188.6082474	128.8111888	166.5731454	2	2
122.00	N S	2936	420600	409700	410200	152.6124819	160.2895149	139.7138965	150.8719644	2	2
123.00	S S	2320	253600	254900	257400	173.6986301	182.0714286	110.9482759	155.5727782	2	2
124.00	A	1904	324600	326700	329200	310.9195402	332.0121951	172.8991597	271.9436317	3	3
125.00	R S	2030	179600	209700	210200	98.68131868	109.21875	103.546798	103.8156222	1	1
126.00	V S	1950	199600	249700	250200	135.7823129	159.044586	128.3076923	141.0448637	1	2
127.00	S A	2640	315600	317700	320200	177.3033708	184.7093023	121.2878788	161.100184	2	2
128.00	P k	1130	143600	145700	148200	135.4716981	145.7	131.1504425	137.4407135	1	1
129.00	J k	2330	240600	242700	245200	163.6734694	172.1276596	105.2360515	147.0123935	2	2
130.00	S D	1630	317600	308600	304000	196.0493827	197.8205128	186.5030675	193.4576543	2	2
131.00	S G	1330	233600	255700	262000	186.88	214.8739496	196.9924812	199.5821436	2	2
132.00	G S	2040	253600	255700	258200	142.4719101	148.6627907	126.5686275	139.2344428	1	1
133.00	T A	2000	199600	209700	210200	131.3157895	129.4444444	105.1	121.9534113	1	1
134.00	RRG	1750	218600	219700	220200	127.0930233	131.5568862	125.8285714	128.1594936	1	1
135.00	HS	2030	224600	244700	264000	123.4065934	139.0340909	130.0492611	130.8299818	1	1

S0	NAME	@48catPLR	mean	LENGTH OF HOSPITAL STAY	TYPE OF MANAGEMENT	ORGAN FAILURE OCCURRENCE	COMPLICATIONS OF ACUTE PANCREATITIS	NEED OF ICU AND HDU STAY	DOR DEATH DAMA DISCHARGE	
69.00	K K	1	1	10	1	0	0	0	discharge	1
70.00	M S	1	1	4	1	0	0	0	discharge	1
71.00	S k	2	2	6	1	1	1	0	discharge	1
72.00	R k	1	1	5	1	0	0	1	DAMA	2
73.00	B k	1	1	5	1	0	1	0	DOR	3
74.00	G S	2	2	16	1	0	0	0	discharge	1
75.00	H k	3	3	24	1	0	0	0	DAMA	2
76.00	k R	1	1	11	1	0	0	0	discharge	1
77.00	k k	1	1	5	1	0	0	0	discharge	1
78.00	K S	1	1	9	1	0	0	0	discharge	1
79.00	L U	2	2	15	1	0	1	1	discharge	1
80.00	M k	2	2	14	1	1	1	0	discharge	1
81.00	N	2	3	11	1	1	1	1	DOR	3
82.00	M K	2	2	24	1	0	0	1	DOR	3
83.00	M D	1	1	9	1	0	0	0	discharge	1
84.00	N	2	2	8	1	1	1	1	DAMA	2
85.00	S K	2	2	19	1	0	1	0	discharge	1
86.00	R K	1	2	12	1	1	1	1	discharge	1
87.00	R S	3	3	21	1	1	1	1	DAMA	2
88.00	S k	2	2	15	1	0	1	1	discharge	1
89.00	N A	2	2	8	1	1	1	0	discharge	1
90.00	N	2	2	21	2	0	1	0	discharge	1
91.00	L	2	2	25	2	0	1	1	discharge	1
92.00	I D T	1	3	18	1	0	0	0	discharge	1
93.00	P	1	1	3	1	0	0	0	DAMA	2
94.00	A S	2	2	9	2	0	1	1	discharge	1
95.00	J p	2	2	10	2	0	1	0	discharge	1
96.00	S D	2	2	9	1	0	1	0	discharge	1
97.00	JS	1	1	3	1	0	1	0	DAMA	2
98.00	M	2	2	10	1	0	0	0	discharge	1
99.00	DR	1	1	4	2	0	1	0	DOR	3
100.00	AS	2	2	6	1	0	0	0	DOR	3
101.00	B S	1	2	14	1	0	1	1	discharge	1
102.00	H P S	1	2	9	1	0	0	0	discharge	1
103.00	R S	1	2	4	1	0	0	0	discharge	1
104.00	S S	2	2	10	1	0	0	0	discharge	1
105.00	B k	1	1	9	1	0	0	0	discharge	1
106.00	S	1	1	9	1	0	0	0	discharge	1
107.00	k k	2	2	9	1	0	0	0	discharge	1
108.00	TPS	1	2	6	1	0	0	0	DOR	3
109.00	V G	1	1	3	1	0	0	0	discharge	1
110.00	SK	1	2	14	1	0	1	0	DOR	3
111.00	S G	2	2	9	1	0	0	0	discharge	1
112.00	B S	1	1	4	1	0	0	0	discharge	1
113.00	R K	1	1	15	1	0	1	0	discharge	1
114.00	M L	2	2	8	1	0	0	0	discharge	1
115.00	A G	2	2	8	1	0	0	0	discharge	1
116.00	S S	1	1	5	1	0	0	0	discharge	1
117.00	V S	1	1	3	1	0	0	0	discharge	1
118.00	U R	1	1	11	1	0	1	1	discharge	1
119.00	R R	2	2	5	1	0	0	0	discharge	1
120.00	P S	1	1	5	1	0	0	0	discharge	1
121.00	P	1	2	7	1	0	0	0	discharge	1
122.00	N S	1	2	9	1	0	0	0	discharge	1
123.00	S S	1	2	8	1	0	0	0	discharge	1
124.00	A	2	2	7	1	0	0	0	discharge	1
125.00	R S	1	1	9	1	0	1	1	discharge	1
126.00	V S	1	1	5	1	0	0	0	discharge	1
127.00	S A	1	2	9	1	0	0	0	discharge	1
128.00	P k	1	1	7	1	0	0	0	discharge	1
129.00	J k	1	1	5	1	0	0	0	discharge	1
130.00	S D	2	2	10	1	0	1	0	discharge	1
131.00	S G	2	2	17	1	0	1	1	discharge	1
132.00	G S	1	1	10	1	0	0	0	discharge	1
133.00	T A	1	1	4	1	0	0	0	discharge	1
134.00	RRG	1	1	5	1	0	0	1	DAMA	2
135.00	HS	1	1	5	1	0	1	0	DOR	3

Email : Examination@bfuhs.ac.in

No. BFUHS/2K21p-TH/ 427 Date 25/11/21

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Shubham Goyal**, a student of your college for **MS General Surgery** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

To study platelet to lymphocyte ratio in predicting the outcome of acute pancreatitis: at admission, 24th and 48th hour.

Supervisor

Dr. Munish Trehan
Assoc. Prof.,
Dept. of Surgery,
DMCH, Ludhiana.

Co-Supervisor

Dr. Jaspal Singh
Prof. & Head,
Dept. of Surgery,
DMCH, Ludhiana.

Dr. Bhavna Garg
Prof.,
Dept. of Pathology,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

135. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
136. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

TO STUDY THE PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION, 24TH AND 48TH HOUR

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MASTER OF SURGERY

In

GENERAL SURGERY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH 2021

DR. SHUBHAM GOYAL

DEPARTMENT OF GENERAL SURGERY
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA
LUDHIANA, PUNJAB-141001

ABSTRACT OF PLAN OF THESIS

Title	TO STUDY PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION, 24TH AND 48TH HOUR"
For the degree of	M.S. General Surgery
Name of the candidate	Dr. Shubham Goyal
Supervisor	Dr. Munish Trehan, Associate Professor, Department of Surgery, DMCH, Ludhiana
Co-supervisor	Dr. Jaspal Singh, Professor & Head, Department of Surgery, DMCH, Ludhiana Dr. Bhavna Garg, Professor, Department of Pathology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Acute pancreatitis is defined as an acute condition presenting with abdominal pain, a three-fold or greater rise in serum levels of pancreatic enzymes amylase or lipase and/or characteristic findings of pancreatic inflammation on imaging methods (including abdominal ultrasonography and computed tomography).
Aim of Study: To study the efficiency of platelet to lymphocyte ratio as marker to predict the outcome of acute pancreatitis.

Materials & methods: This prospective study will include patients presenting with acute pancreatitis in Dayanand medical college and hospital between 1st march,2021 to 28th feb,2022. Platelet to lymphocyte ratio will be calculated as ratio of absolute platelet count to absolute lymphocyte count at the time of presentation to hospital and at 24th and 48th hour of presentation to hospital. Mean of PLR value at time of presentation, 24th and 48th hour of presentation to hospital will be calculated. Patients will be divided into 3 groups according to mean platelet to lymphocyte ratio of < 150 as ist group, 150-300 as 2nd group and > 300 as 3rd group and length of hospital stay, need of ICU/HDU stay, DOR/death/DAMA/discharge, type of management whether (medical, PCD, surgical), organ failure occurrence/MODS, complications of acute pancreatitis will be compared between the groups. The results will be statistically analysed.

Clinical Significance: By knowing PLR value at the time of admission, 24th and 48th hour and by predicting the possible outcome, the management can be more focused.

Keywords: Acute pancreatitis (AP), Platelet to Lymphocyte ratio (PLR)

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO STUDY PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION, 24TH AND 48TH HOUR"** exist in the Department of Surgery, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate himself under our supervision and guidance.

Supervisor

Dr. Munish Trehan
Associate Professor
Department of General Surgery
DMCH, Ludhiana

Co-Supervisors

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Head of the Department

Dr. Jaspal Singh
Professor & Head
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Place: _____

Date: _____

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Shubham Goyal
Department	General Surgery
Topic of Thesis	TO STUDY THE PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION,24TH AND 48TH HOUR"
Likely date of appearing in PG examination	MAY 2023
Date of enrollment	26th July, 2020
Name of Head of Department	Dr. Jaspal Singh
1) Supervisor **2) Co-Supervisor**	Dr. Munish Trehan Dr. Jaspal Singh Dr. Bhavna Garg
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairman
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	DR. SHUBHAM GOYAL STREET NO. 7 INDRA BASTI DISTRICT SANGRUR, PUNJAB
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA
3	COURSE OF STUDY AND SUBJECT	M.S GENERAL SURGERY
4	DATE OF ADMISSION OF COURSE	26TH JULY 2020
5	TITLE OF THE TOPIC	"TO STUDY THE PLATELET TO LYMPHOCYTE RATIO IN PREDICTING OUTCOME OF ACUTE PANCREATITIS AT ADMISSION, 24TH AND 48TH HOUR"

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Acute pancreatitis is defined as an acute condition presenting with abdominal pain, a three-fold or greater rise in serum levels of pancreatic enzymes amylase or lipase and/or characteristic findings of pancreatic inflammation on imaging methods (including abdominal ultrasonography and computed tomography [1]. Patients with high platelet to lymphocyte values have increased prevalence of icu admissions, increased timespan of hospital stay in survivors and higher mortality therefore, higher PLR values are associated with poor prognosis in acute pancreatitis [1]. Acute pancreatitis can present as clinically from no symptoms to SIRS, persistent organ failure and death. The clinical signs of acute pancreatitis can't exactly anticipate the severity of acute pancreatitis [2]. In AP mortality that occurs early is due to SIRS, resulting in MODS, while mortality that occurs delayed is generally due to sepsis and its complications [3]. Therefore, it important to diagnose and treat the disease early and to do the precise risk assessment to divide patients into high or low risk categories, and to admit patients with organ dysfunction to the intensive care units [4]. Various scoring methods were developed to early acknowledge acute pancreatitis and to conclude the plan of treatment. The most common scoring methods are the acute physiologic assessment and chronic health evaluation (apache) and ranson criteria [5]. These scoring systems use many variables which are laborious to obtain in low resource settings and are cumbersome [1]. In recent time, we still need simple, effective and rapid markers to forecast the severity of acute pancreatitis prognosis [6]. These markers can be acquired from economical laboratory tests such as neutrophil to lymphocyte ratio(nlr), red cell distribution width(rdw), mean platelet volume and platelet to lymphocyte ratio [6]. The platelet activating factor released for the most part from vascular endothelium is pro inflammatory mediator have primary importance in inflammation in AP and cause localized tissue damage and bleeding. PAF start up the platelet and neutrophils,

increases the capillary permeability [7]. Platelets are being incite by contact with artery surface and are a source of inflammatory mediators[8]. The platelet to lymphocyte ratio has come out as an informative marker which disclose shifts in platelet to lymphocyte counts due to acute inflammatory and prothrombotic states. Platelet to lymphocyte ratio which can be forecast by combined blood cell counts and their ratios has been exhaustively examined in neoplastic diseases accompanied by immune suppression and thrombosis [9]. Platelet counts match up with platelet reactivity and volume and may designate autoimmune disease activity, response to anti-inflammatory therapies, and presence of various comorbidities[10-12] In vitro and vivo studies have clear up the origin of activated circulating platelets, which exhibit countless membrane receptors and release multiple biologically active substances from their granules that can cope cellular interactions and put up to immune, inflammatory and thrombotic diseases[13-15]. Circulating platelets can interact with erythrocytes, neutrophils and lymphocytes in the vessel lumen at the sites of vascular damage [16,17]. The interaction of platelets with t lymphocytes, mediated by the p-selectin bring down the lymphocyte proliferation, resulting in a decrease in proinflammatory cytokines, such as interferon- alpha (IFN-ALPHA), tumour necrosis factor-alpha (TNF ALPHA) and interleukin (IL-17), and an increase in anti-inflammatory cytokines such as IL-10[18]. Transient lymphopenia and enormity of stress- induced hypercortisolaemia with subsequent release of platelets into blood stream impact the degree of elevation of plr across countless proinflammatory and prothrombotic disease states. Such a non-specific mechanism of PLR elevation can be impeded by intensified platelet destruction or consumption at the sites of immune inflammation and thrombosis, compelling cross- checks of all blood cells counts and other inflammatory and immune markers [19].

The aim of present study will be to search the efficiency of platelet to lymphocyte ratio in predicting the severity of acute pancreatitis.

6.2 REVIEW OF LITERATURE

The triggering mechanism that causes AP is the activation of trypsinogen in the pancreas. Active trypsin also activates complement system and kallikrein-kinin cascade along with coagulation and fibrinolysis [1]. It has been noted that severe COVID-19 patients have elevated platelet to lymphocyte levels on admission due to rapid involvement of inflammatory processes in COVID-19. This put forward the possible use of this inflammatory marker to determine the prognosis of COVID-19 patients especially in resource limited settings[20,21]. High platelet to lymphocyte ratio along with elevated platelet count, is possively useful in diagnosing some systemic vascular diseases such as giant cell arteritis (Arme yuri Gasparyan et al) [9]. demir et al, investigated the relationship between high platelet to lymphocyte ratio and mortality in patients with acute cardiogenic pulmonary edema and ascertain that high PLR group had notably higher mortality rate than other groups [22] Cho sk et al, investigated the relationship between NLR and PLR in outcome in patients of acute pancreatitis [23]. Changes in platelet number and coagulation anomalies have been determined to be associated with disease severity in acute pancreatitis [24].

Reddy CS et al (2019) conducted study on 256 patients, who fulfil all the inclusion criteria and were lacking exclusion criteria's and were stratified into tertiles depends on the PLR value at admission.

85(33.2%) patients had a PLR of less than or equal to 150 (1st tertile), 88 (34.4%) patients had a PLR between 150-300 (2nd tertile) and 83 (32.4%) patients had a PLR greater than or equal to 300 (3rd tertile). The incidence among the three groups was identical, with no statistical difference. The mean of the age group in the 1st tertile was 46.2, which was less than the mean age of 2nd tertile, which was 48.6 and the mean age group of the 3rd tertile was higher when compared to the other two tertile of the group. Patients of higher age group had greater PLR values [1].

The risk factors such as smoking, alcohol abuse and diabetes mellitus had a greater incidence in the patients presenting with the 2nd tertile

value of PLR, showing an incidence of 80.6%, 65% and 30% respectively. Whereas, hypertension was more common in the 3rd tertile, with an incidence of 44.5%. The mean length of the hospital stay was found to be 5 days in first tertile, 11 days in the second tertile and 18 days in the third tertile which put forward longer stay in the third tertile group, which was statistically remarkable. The requirement of ICU admission was found in six patients (7% of patients) in the first tertile, in 26 patients (29.5% of patients) in the second tertile and in 59 patients (71% of patients) in the third tertile which put forward greater incidence of ICU admissions in the third tertile, i.e., 71% of the total patients in the third tertile, when compared to the other two groups. The mortality rate was found to be 1.1% (1 patient) in the first tertile, 9.1% (8 patients) in the second tertile and 28.9% (24 patients) in the third tertile which put forward the great incidence of mortality in the third tertile, i.e., 28.9% of the total patients in the third tertile, when compared with the other two groups.

Statistically, higher PLR was associated with notable change in the outcomes, that is greater length of hospital stay, greater requirement of ICU admission and increased incidence of mortality [1].

Gokden Y et al (2020) studied total of 140 patients of AP which consists of 76 females (54.3%) and 64 males (45.7%) with an mean age of 52+_ 15.5 and a total of 49 individuals were encompass in healthy control (HC) group consisting of 30 females (61,2 %) and 19 males (38.8%) with mean age of 46+- 12.9.[18]

There was statistically difference with regard to age, neutrophil, lymphocyte and PLR levels between AP and HC groups ($p = 0.007$; $p<0.001$; $p<0.001$ and $p<0.001$, respectively.)

When correlation was performed according age, age variable was statistically different between the groups (i.e., covariance analysis), neutrophil, lymphocyte and PLR levels were still different between the groups ($p<0.05$). Neutrophil and PLR levels in AP group were statistically significantly higher compared to HC group ($p<0.001$). No statistically significant difference was determined between AP group and HC group with regard to PLT and MPV levels($p> 0.05$). AP patients

were compared with regard to MPV and PLR by grouping under biliary and non-biliary groups according to their etiology, ≤150 mg/L and >150 mg/L groups according to CRP levels at the 48th hour, as mild and severe AP patients according to Ranson criteria, and as mild-moderate and severe AP patients according to revised Atlanta criteria.

MPV was found to be statistically significantly lower in the AP group with >150 mg/L CRP level compared to the AP group with ≤150 mg/L CRP level (p=0.046).

PLR was found to be statistically significantly higher in AP group with >150 mg/L CRP level (p=0.014). MPV was found to be statistically significantly lower in non-biliary AP group compared to biliary AP group (p=0.034), and no statistically significant difference was determined between PLR variables of both groups (p>0.05).No statistically significant difference was determined between mild AP group and severe AP group with regard to MPV and PLR according to Ranson criteria (p>0.05).PLR was determined to be statistically significantly higher in severe AP group compared to mild-moderate AP group according to revised Atlanta classification (p=0.023), and no statistically significant difference was noted between MPV levels of both groups (p>0.05). According to their Balthazar score, AP patients was grouped as mild, moderate and severe patients, and compared with regard to MPV and PLR.

No statistically significant difference was determined among AP patients grouped as mild, moderate and severe according to Balthazar score with regard to MPV and PLR(p>0.05).

In AP diagnosis for testing the usability of PLR as a determinant test and determining diagnostic test performance parameters and applicable cut-points belonging to parameters used in activity scoring, AP/HC, CRP and Revised Atlanta criteria were determined as state variables and ROC analysis was performed.

In the result of ROC analysis, it was determined for PLR in AP and HC that the diagnostic threshold value was “123” for determining sick individuals, diagnostic threshold value was “153” for determining for >150 CRP at 48th hour, and the diagnostic threshold value was “151”

for severe AP according to Revised Atlanta criteria. By using determined threshold values for PLR, Sensitivity, Specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV),
Accuracy and Standard Error (SE), and Area under the curve AUC) with P value demonstrated under 95% Confidence Interval (CI) in Figure 1,2,3.

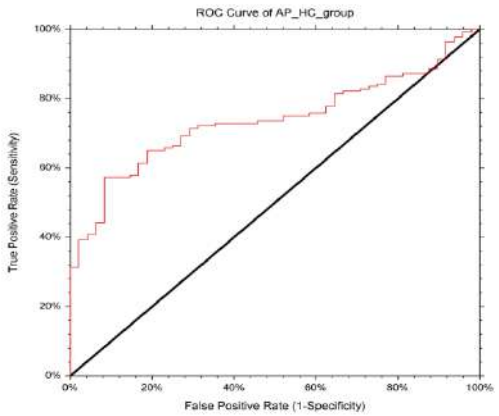

Figure 1. ROC curve graph of PLR variable according to AP-HC group

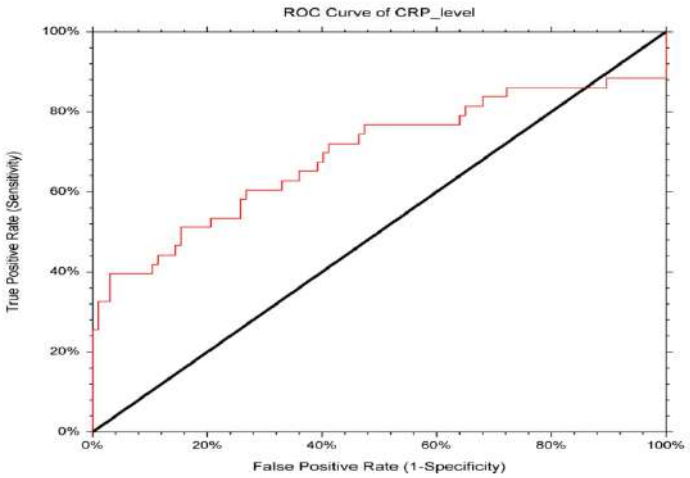

Figure 2. ROC curve graph of PLR variable according to CRP level

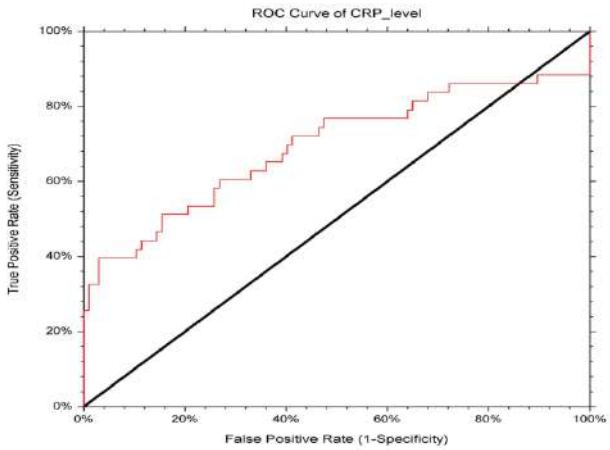

Figure 3. ROC curve graph of PLR variable according to Revised Atlanta criteria

The relation between PLR, MPV and Hospitalization Period variables in AP patients was examined with Spearman correlation analysis. In non-biliary AP group, a statistically significant positive weak correlation was determined between hospitalization period and PLR (r=0.239; p=0.049) and a statistically significant negative weak correlation was determined between MPV and PLR (r=-0.256; p=0.035), and no statistically significant correlation was determined between hospitalization period and MPV (p>0.05) No statistically significant correlation was determined between PLR, MPV and hospitalization periods in the biliary acute pancreatitis patient group (p>0.05) [25].

6.3 AIMS AND OBJECTIVES OF THE STUDY

To study the efficiency of platelet to lymphocyte ratio as marker to predict the outcome of acute pancreatitis.

7. MATERIALS AND METHODS

7.1 Source of Data

This study would be prospective study, after approval from institutional ethics committee (IEC). Written, informed consent would be obtained from all patients.

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA-

Patients presenting to the hospital with diagnosis of acute pancreatitis.

EXCLUSION CRITERIA-

Patients would be excluded from this study if they had any of the following:

1. Pancreatitis developed as a result of trauma.
2. Patients with age less than 18 years of age.
3. Patients with diagnosed case of hematological disorders, cancers and patients on chemotherapy.
4. Patients not giving consent to participate in study.

This prospective study will include patients presenting with acute pancreatitis in Dayanand medical college and hospital between 1st march 2021 to 28th feb 2022. At least two of the following three symptoms will be sought-out criteria for Acute Pancreatitis diagnosis: acute condition presenting with abdominal pain, a threefold or greater rise in serum levels of pancreatic enzymes amylase or lipase and characteristic findings of pancreatic inflammation on imaging methods (including abdominal sonography and computed tomography). Patients with pancreatitis developed as result of trauma, patients with age less than 18 years of age, patients with haematological disorders, cancers and patients on chemotherapy, patients not giving consent to participate in study will be excluded from study. Demographic characteristics, vital signs and laboratory parameters (white blood count, neutrophil count, lymphocyte count, haemoglobin level, platelet count, Red cell distribution width, mean platelet volume, haematocrit) will be assessed and will be recorded in all patients. Platelet to

lymphocyte ratio will be calculated as ratio of absolute platelet count to absolute lymphocyte count at the time of presentation to hospital and at 24th and 48th hour of presentation to hospital. Mean of PLR value at time of presentation, 24th and 48th hour of presentation to hospital will be calculated. we will divide the patients diagnosed with acute pancreatitis into 3 groups according to mean platelet to lymphocyte ratio of <150 as ist group, a platelet to lymphocyte ratio of 150-300 as 2nd group and >300 as 3rd group and length of hospital stay and need of ICU/HDU stay, DOR/death/DAMA/discharge, type of management, organ failure occurrence as defined by [shock(systolic blood pressure <90mm hg), pulmonary insufficiency (pao2 < 60 mm hg), renal failure(creatinine level > 2 mg/dl after fluid resuscitation), gastrointestinal bleeding (>500 ml/24 hr)] according to the ATLANTA criteria for acute pancreatitis and MODS(multiple organ dysfunction syndrome), complications of acute pancreatitis like peripancreatic fluid collections, pancreatic necrosis and infected necrosis, pancreatic pseudocyst, pancreatic ascites and pancreaticopleural fistulas, vascular complications, pancreatocutaneous fistula will be compared between the groups. The results will be statistically analysed.

Duration of study: 1 year (from 1st march 2021 to 28th feb 2022)

OUTCOME

Patients fulfilling the inclusion criteria will be evaluated prospectively and final outcome will be noted in view of:

1) length of hospital stay
2) Need of Icu stay/ hdu stay
3) DOR/death/DAMA/discharge
4) Type of management
5) Organ failure occurrence/MODS
6) Complications of acute pancreatitis

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done

using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Patients presenting included in the study will have to undergo haematological, biochemical or radiological investigations. For this patient included in the study will be informed regarding their inclusion and written informed consent will be taken for all the patients included in the study

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached here with

LIST OF REFERENCES

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PROFORMA

SR. NO. MRD NO.

NAME:	AGE/SEX:
ADDRESS:	OCCUPATION:
DATE OF ADMISSION:	DATE OF DISCHARGE:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST MEDICAL/ SURGICAL HISTORY:

PERSONAL HISTORY:

Marital status..... Dietary habits..... Smoking.....

Alcohol..... Substance abuse..... Other.....

FAMILY HISTORY:

BLOOD INVESTIGATIONS:

ON ADMISSION:	AT 24TH HOUR:	AT 48TH HOUR
PLATELET COUNT:	PLATELET COUNT:	PLATELET COUNT:
LYMPHOCYTE COUNT:	LYMPHOCYTE COUNT:	LYMPHOCYTE COUNT:
PLR RATIO:	PLR RATIO:	PLR RATIO:

DATE:					
HB					
TLC					
NEUTROPHIL COUNT					
MEAN PLATELET VOLUME					
HEMATOCRIT					
RDW					
BLOOD UREA					
SERUM CREATININE					
SERUM AMYLASE					
SERUM LIPASE					
SERUM CALCIUM					

LFT :

DATE:					
BILIRUBIN(T)					
BILIRUBIN(D)					
SGOT					
SGPT					
ALP					
TOTAL PROTEINS					
ALBUMIN					

ABG ANALYSIS:

DATE:					
pH					
Pco2					
Po2					
lactate					
Hct					
Hco3-					
Base excess					

GENERAL PHYSICAL EXAMINATION:

Appearance……. Sensorium…….

Vital signs: HR…… RR……. BP……. TEMP…….

Pallor……. Jaundice……. Cyanosis……. Edema…….

JVP……. Lymphadenopathy……. Clubbing…….

spo2(room air) ……. spo2(on oxygen) …….

GCS: E V M

Pupillary size (L) ……. (R) ……. Reaction(L) ……. (R) …….

ANTHROPOMETEY:

weight(kg) ……. height(m) ……. BMI…….

Build……. Anemia……. vitamin deficiency…….

nutrition status…….

RADIOLOGICAL INVESTIGATIONS (USG/CT/MRI):

ABG ANALYSIS:

DATE:					
pH					
Pco2					
Po2					
lactate					
Hct					
Hco3-					
Base excess					

GENERAL PHYSICAL EXAMINATION:

Appearance……. Sensorium…….

Vital signs: HR…… RR……. BP……. TEMP…….

Pallor……. Jaundice……. Cyanosis……. Edema…….

JVP……. Lymphadenopathy……. Clubbing…….

spo2(room air) ……. spo2(on oxygen) …….

GCS: E V M

Pupillary size (L) ……. (R) ……. Reaction(L) ……. (R) …….

ANTHROPOMETEY:

weight(kg) ……. height(m) ……. BMI…….

Build……. Anemia……. vitamin deficiency…….

nutrition status…….

RADIOLOGICAL INVESTIGATIONS (USG/CT/MRI):

SYSTEMIC EXAMINATION:

ABDOMEN:

RESPIRATORY:

CVS:

CNS:

ETIOLOGY OF ACUTE PANCREATITIS:

FINAL OUTCOME:

LENGTH OF HOSPITAL STAY	
NEED OF ICU/HDU STAY	
DOR/DEATH/DAMA/discharge	
TYPE OF MANAGEMENT (MEDICAL/PCD/SURGICAL)	
ORGAN FAILURE OCCURRENCE/ MODS	
COMPLICATIONS OF ACUTE PANCREATITIS	

SIGNATURE OF CANDIDATE: **SIGNATURE OF SUPERVISOR:**

INFORMED PATIENT CONSENT

Date: _____

I..... D/o, W/o, S/o..... am willing to participate in the study **"TO STUDY PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION, 24TH AND 48TH HOUR"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"TO STUDY PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION, 24TH AND 48TH HOUR"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"TO STUDY PLATELET TO LYMPHOCYTE RATIO IN PREDICTING THE OUTCOME OF ACUTE PANCREATITIS: AT ADMISSION, 24TH AND 48TH HOUR" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

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ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

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ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

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ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ